California MEDICINE

MAY 1949

BLOOD TRANSFUSIONS AND THE Rh FACTOR, Paul G. Hattersley, San Francisco 379

HEAD RETRACTION REFLEX, Robert Wartenberg, San Francisco 382

HEART DISEASE AS A COMPLICATION OF PREGNANCY, John J. Sampson, San Francisco 383

SURGICAL TREATMENT OF PATENT DUCTUS ARTERIOSUS, John C. Jones, Los Angeles 391

SUBSTERNAL GOITER AND PULMONARY EMPHYSEMA, Edward Phillips, Los Angeles, and Samuel A. Levine, Boston 394

RESULTS OF MEDICAL AND SURGICAL MANAGEMENT IN TEN CASES OF CONGENITAL MEGACOLON, Louise A. Yeazell, H. Glenn Bell, San Francisco 398

DYSPLASIA OF THE HIP: A FACTOR IN THE DEVELOPMENT OF STATIC OSTEOARTHROPATHY, Joseph Levitin, San Francisco 401

CLINICAL APPLICATION OF ELECTROKYMOGRAPHY, Frederick G. Gillick and William F. Reynolds, San Francisco 407

THE PAPANICOLAOU-TRAUT METHOD OF CANCER DIAGNOSIS, Its Use as a Routine Pathologic Laboratory Procedure, Stuart Lindsay, San Francisco 413

CONTAGIOUS ECTHYMA IN MAN, Earl H. Gray, Woodland 417

Case Reports:

Temporal Arteritis, Thomas H. Lambert, La Jolla, San Diego 419

Charcot's Joint, with Unusual Features, in a Diabetic Patient, Ira H. Wilson, Charles H. McIntyre, and H. K. Albertson, San Diego 420

Ruptures of the Umbilical Cord with a Case of Intrapartum Rupture of All Three Vessels, H. E. Bowles and R. D. McKee, Honolulu 422

California Cancer Commission Studies:

Tumors of the Brain and Spinal Cord, Howard C. Naffziger, San Francisco 424

EDITORIALS 427

LETTERS TO THE EDITOR 428

CALIFORNIA MEDICAL ASSOCIATION 429

NEWS AND NOTES 439

INFORMATION 441

BOOK REVIEWS 443

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Blood Transfusions and the Rh Factor

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SUMMARY

When Rh-negative persons are given transfusions of Rh-positive blood, more than 50 per cent are sensitized to the Rh_0 factor. Such sensitization of female children may be the cause of hemolytic disease in their offspring many years later, while severe hemolytic reactions may follow a second transfusion of Rh-positive blood in either sex.

The gross hemolysis of transfused blood may be entirely asymptomatic, however. In one case a pint of blood was completely hemolyzed within two hours without producing symptoms. The only signs were hemoglobinuria, low grade jaundice, urobilinogenuria and a rising Rh antibody titer. The patient had previously been sensitized by a single pint of Rh-positive blood.

The dangers of Rh sensitization can be avoided by routine Rh typing of all prospective recipients of transfusion, whether male or female, and by giving only Rh-negative blood to those who are Rh-negative.

SINCE 1940, when Landsteiner and Wiener[5] first described the newly discovered "Rhesus factor" in human erythrocytes, certain basic facts regarding this factor have become so well established and so generally known as to require only the briefest review:

1. The Rh and Hr factors are a group of antigenic substances which occur in various combinations in the red blood cells of all human beings.

2. These factors are hereditary characteristics determined by corresponding genes, each factor being a simple mendelian characteristic, dominant over its absence.

3. The Rh factors, being antigenic substances, are capable of stimulating the production of type-specific antibodies when cells containing these antigens are injected into receptive individuals or into suitable experimental animals. Antibodies corresponding to each of the known Rh and Hr antigens have been found in the blood of sensitized patients. Sera containing these antibodies are utilized in typing human erythrocytes.

4. A single member of this group of antigens, the Rh_0 (or D of Fisher) has proven by far the most important, probably 99 per cent of all Rh sensitizations occurring among the 15 per cent of persons whose cells lack this antigen. Because of this fact, the anti-Rh_0 (anti-D) serum is known as the routine or diagnostic Rh-typing serum, and patients are classified as Rh-positive or Rh-negative on the basis of the reaction of their cells in this serum.

5. Sensitization of Rh-negative patients to the Rh factors occurs in two ways: First, by transfusion with Rh-positive blood; and, second, by repeated pregnancy and delivery of Rh-positive infants. In the latter case, the fetus receives the dominant Rh-positive gene from the Rh-positive father. It is probable that during the latter months of pregnancy, when the placental villi become progressively thinner, with less of a barrier separating fetal and maternal circulations, some fetal red blood cells, containing the Rh antigen, pass into the maternal circulation, where they stimulate the formation

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Read as a part of a Symposium on Hematology before a joint meeting of the Sections on General Medicine and Public Health at the 77th Annual Session of the California Medical Association in San Francisco, April 11-14, 1948.

of Rh antibodies. These antibodies in turn diffuse back into the fetal blood, where they destroy the fetal red blood cells and tissues, causing the disease known as erythroblastosis fetalis, or hemolytic disease of the newborn.

These are the general facts concerning Rh, facts which concern primarily the obstetrician, the pediatrician and the clinical pathologist. There is one aspect of the subject, however, with which every practicing physician should be acquainted, in this day of increasing use of blood transfusions. I refer to the serious hazard of sensitization of the Rh-negative patient by transfusion.

It has been estimated that from 1 in 25 to 1 in 50[9, 11] Rh-negative women who give birth to Rh-positive infants become sensitized to the Rh factor. It was for some time considered that the remainder were incapable of producing Rh antibodies, and that the incidence of sensitization by transfusion was very similar to that by pregnancy.

Some relatively recent work, however, has demonstrated that the *majority* of Rh-negative persons develop Rh antibodies following repeated transfusions of Rh-positive blood. Diamond and his coworkers[1, 2] studied a group of several hundred Rh-negative service men who had received blood transfusions overseas, and found that over 50 per cent of them had demonstrable Rh antibodies in their sera. These observations have been corroborated in smaller series elsewhere.[4, 8] Diamond[1] likewise found Rh antibodies in the sera of a majority of unmarried Rh-negative women who had received blood transfusions; and among a small series of women who had experienced both transfusions and pregnancies, nearly all showed evidence of sensitization. More recently Diamond[5] and Wiener and Sonn-Gordon[12] succeeded in sensitizing a very large percentage of Rh-negative volunteers by repeated injections of Rh-positive blood.

That sensitization by transfusion may be responsible for severe hemolytic disease in infants born to such women has, of course, been well established. The following is a characteristic case:

CASE REPORT

A 25-year-old housewife was referred because of the death of her first two infants. The first was born at term and appeared normal, but rapidly developed intense jaundice and died on the second day. The second pregnancy, two years later, resulted in a stillborn hydropic fetus. Pathologic examinations on both infants revealed the characteristic picture of severe erythroblastosis fetalis.

Laboratory tests showed the patient's blood to be group A, Rh-negative, and the serum contained hyperimmune antibodies of Rh_0 specificity to a titer of 512. Her husband was Rh-positive, type Rh_1, Hr′-negative and Hr_0-negative (CDe/CDe). As these reactions indicated that he was homozygous Rh-positive, and that any children he might have would likewise be Rh-positive, it was necessary to advise this young couple that their chance for having children of their own was extremely poor.

Careful history revealed that as a girl, this patient had received a series of transfusions for anemia associated with nephritis.

Cases of this sort are unfortunately not particularly rare. Levine and Waller[6] have reported a series of 19, some of the patients having been sensitized by transfusion in early childhood. There is much evidence that Rh sensitization, once it is gained, is never lost. Even though the antibody may disappear from the blood, subsequent exposure to the Rh antigen elicits its prompt reappearance.

It is also rather common to encounter women who have had one or more perfectly normal children, but who then, following a transfusion, have delivered severely affected infants. It is becoming increasingly recognized that in order to prevent such tragedies, the Rh-negative female who has not passed childbearing age should never receive transfusions of Rh-positive blood—even in infancy.

On the other hand it is certainly less generally realized that even among male patients transfusion without preliminary Rh typing may be very dangerous. The picture of the acute transfusion reaction in its classic form is all too well known: The sudden chill with substernal or lumbar pain, the fall in blood pressure, often to shock levels, and the subsequent urinary suppression and uremia, often terminating fatally. Such reactions are fortunately rather rare, usually following transfusion with blood of incompatible major blood group. The fact that transfusion reactions due to Rh incompatibility are only rather rarely reported, and are usually among postpartum women, has been responsible for the common belief that Rh sensitization by transfusion is an unusual occurrence, and that it is only among the female population that routine Rh typing before transfusion should be practiced.

It has been pointed out by various workers,[3, 7] however, that hemolysis of transfused cells may occur without the characteristic signs and symptoms. It has been suggested that the lack of symptoms is due to the fact that the transfused cells are hemolyzed slowly in such cases, rather than immediately, as they presumably are when, for instance, group A blood is given to a group O patient. That asymptomatic hemolytic reactions do occur, but that even in such cases destruction of the hemolyzed blood may be very rapid, is illustrated by the following case:

CASE REPORT

A 48-year-old male Army officer received his first transfusion when injured in the Philippines. Six months later he was brought to a San Francisco hospital for surgical repair of his injuries, and while in the operating room he received 500 cc. of two-day-old whole bank blood drawn into acid citrate dextrose solution. Due to a clerical error in the laboratory, this patient had been recorded as Rh-positive, although he actually had not been Rh-typed at all. His blood was cross-matched with that of a group A Rh-positive donor, saline-suspended cells of patient and donor each being incubated with the serum of the other for 30 minutes at 37° C., and the tubes centrifuged. Since the cross-match showed no agglutination, the blood was considered compatible, and was sent to the operating room. The patient, who was conscious throughout the transfusion, did not complain of chill or discomfort, the blood pressure remained steady, and during the subsequent hours there was no elevation of temperature.

In the meantime the error had been discovered, and it was determined that the patient was actually Rh-negative. Another blood specimen was drawn immediately after his return from the operating room, and the Rh typing confirmed. In examining this specimen, however, two unexpected discoveries were made: (1) The patient's serum was brownish and appeared to contain not only an increase in bilirubin, but free hemoglobin as well; (2) although it had been less than two hours since he had received a pint of Rh-positive blood, *no Rh-positive cells could be found in the specimen.** It appeared that the transfused cells had been completely hemolyzed. The pre-transfusion blood specimen that had been taken from the patient then was tested for Rh antibodies, and was found to have hyperimmune antibodies of Rh_0 specificity to a titer of 64, and of Rh′ specificity to a titer of 8. There was no activity in saline.

During the ensuing 24 hours the patient passed brownish urine which gave a positive reaction to a chemical test for hemoglobin and which contained large amounts of urobilinogen. Although there was only the faintest discernible trace of icterus of the sclerae, the serum bilirubin rose to 3.0 mg. per 100 cc., the one-minute test being negative. Hemoglobinuria and bilirubinemia soon cleared, there was no suppression of urinary output, and during the three months that the patient was followed at this hospital, blood urea did not rise above normal levels. At the end of that time the only demonstrable effect of the transfusion reaction was his Rh-antibody titer which had risen to 4,000.

There are several lessons to be learned from this case, other than the obvious importance of keeping clerical errors out of the transfusion section: (1) The patient had been sensitized by a single transfusion six months before. Although sensitization is usually said to occur only after several transfusions, the author not infrequently has noted antibodies in the serum of patients who have received only one or two transfusions and who could not have been sensitized by pregnancy. (2) Although the patient's serum contained definite Rh antibodies, the routine tube cross-match with the Rh-positive bank blood did not reveal the incompatibility. This was due to the fact that the cross-match technique, although one widely used, was not designed to detect the *hyperimmune* or *blocking* type of Rh antibodies which usually occur in the blood of individuals sensitized by transfusions and which do not agglutinate saline-suspended cells. (3) Although the erythrocytes of an entire pint of blood apparently were hemolyzed within two hours, there were none of the symptoms usually associated with acute hemolytic reactions, and the only signs—the hemoglobinuria and icterus—were so minimal as to be missed entirely by the clinician until pointed out to him by a member of the transfusion section.

There is much reason to believe that asymptomatic transfusion reactions of this sort are not uncommon. Although the practice at Stanford University Hospital of routinely Rh-typing the blood of all patients who are to be given transfusion has obviated such reactions, the author has observed a few patients at other hospitals who hemolyzed one or more pints of blood with little or nothing in the way of symptoms. Likewise the author has seen a number of patients who had received as many as 30 or 40 transfusions elsewhere, whose serum contained a high titer of Rh antibodies, but who denied any symptoms of reaction. Surely many of them must have hemolyzed some of the transfused blood asymptomatically, just as did the patient in the case reported.

It is not surprising that such events should rarely be reported, since, in the absence of the characteristic chill, the average clinician does not feel it necessary to search for the less obvious signs of hemolysis. If such search were made, however, it probably would be discovered that, among Rh-negative patients who have received blood previously, apparently uneventful transfusions of Rh-positive blood are not infrequently followed by hemoglobinemia, hemoglobinuria, low-grade icterus and urobilinogenuria, and that such transfusions do not elevate the hemoglobin as they should.

In spite of their apparent innocuousness, such asymptomatic hemolytic reactions must not be dismissed as of no importance. In the first place, transfusions of incompatible blood are at best a waste of blood and of no help to the patient. On that basis alone they should be avoided. Secondly, it has yet to be determined that such reactions do not injure the patient. Only time and thorough study will determine whether there is permanent renal or hepatic damage in such patients. Finally, and most important, there is as yet no way of predicting which patients will hemolyze transfused blood without symptoms and which will die in shock or in renal failure. In view of these facts, it is the opinion of many, including the author, that transfusions of Rh-incompatible blood should be avoided, even in the male patient. The only way in which this can be done is to institute Rh-typing as a routine pre-transfusion procedure and to maintain a source of Rh-negative blood for the Rh-negative patients.

That this is not universally the practice, even with female patients, is due more to a lack of appreciation of its importance by the average physician than to any lack of facilities. Rh-typing techniques have been so simplified that they can be carried out reliably in the smallest laboratories and by the least trained personnel. Reliable Rh antisera are becoming increasingly available and the modern blood banks are prepared to supply Rh-negative blood on call. These facilities will not be properly utilized, however, or extended to smaller communities, where the big city blood bank is not available, until the medical profession becomes more fully aware of the potential for trouble in the Rh-incompatible blood transfusion.

*The technique used was one utilizing the blocking antibody in bovine albumin. The failure to demonstrate Rh-positive cells could not have been due to "blocking" of them by antibodies in the patient's serum.

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Head Retraction Reflex

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ON BRISK stretching, every striated muscle reacts with brisk contraction. This reaction constitutes the deep muscle reflex. These reflexes are usually designated in textbooks by such terms as tendon, bone, periosteal, and osteoperiosteal reflexes. These names, however, are misleading, since the tendon, the periosteum, and the bone are simply places on which the blow of the reflex hammer is applied in order to evoke sudden stretching of the muscle. Such stimulation in itself does not initiate the reflex. In elicitation of the reflex, it is solely the stretching of the muscle fibers *en masse* that allows it to come into action. The term "deep muscle reflexes" is, therefore, appropriate. It follows, then, that it is not logical to designate each reflex according to the point of elicitation, since these points may be different and varied. It is far better to apply the names of the involved muscles—which when stretched respond and contract forthwith—than to associate the reflexes with the points of their elicitation, or to give them proper names. Monrad-Krohn in the eighth edition of his popular work, "The Clinical Examination of the Nervous System," uses the terms "glabella reflex (supraorbital periosteal reflex)" and "radialis periosteal reflex (supinator jerk)." How much simpler to say "orbicularis oculi reflex" and "brachioradial reflex"! Why use proper names as Hoffmann, Troemner, Bechterew, when the reflex named for them is, physiologically, a "finger flexor reflex"?

Some muscles react to stretching with contraction more easily than do others. Some are so located that it is difficult to stretch them briskly and effectively, or if they do contract, the contraction and the movement effected thereby is hardly discernible.

It is of paramount importance for the understanding of the deep muscle reflexes and for the evaluation of their manifestations to realize that in some muscles the threshold for contraction on stretching is so high, the visible manifestation of this contraction so delicate, that in normal it is hardly discernible on gross inspection and palpation. This is true, for instance, of the deep muscle reflex of the plantar muscles, the flexors of the toes. The so-called Rossolimo reflex is nothing else than this deep plantar muscle reflex; it is therefore not in itself pathologic, but is only a pathologic exaggeration of a normal but latent deep muscle reflex.

The same applies to the reflex of the muscles in the nape of the neck, which retract the head. This reflex cannot be elicited in normal persons but becomes manifest when the corresponding reflex arc is released from pyramidal control, as in cases of bilateral supracervical affection of the pyramidal tract. To elicit a stretch reflex of these muscles, the patient must keep his neck muscles loose and his head slightly bent forward. The examiner applies a blow downward to the middle of the upper lip in order to effect a brisk bending of the head. When this reflex is positive, the patient answers with a quick retraction of his head. The deep muscles in the nape of the neck, the head retractors, react to sudden stretching in the same way as any other muscles. This is the head retractors' reflex, or, for the sake of euphony, head retraction reflex. This reflex has been found positive in the presence of lesions of the pyramidal tracts above the cervical cords—in diffuse brain lesions, as cerebral arteriosclerosis, hypertensive encephalopathy, cerebral lues. It is further positive in the presence of all spinal lesions which have a tendency to creep upward and to affect the pyramidal tracts of the cerebral hemispheres, for instance in amyotrophic lateral sclerosis, lateral sclerosis, dorsolateral sclerosis. This reflex may thus indicate whether a spinal lesion has transgressed the boundaries of the spinal cord and has affected the pyramidal tracts above the cervical cord.

Physiologically the head retraction reflex belongs to the syndrome of decerebrate rigidity in which the retraction of the head may be very pronounced. It is an expression of the mildest, subclinical form of such decerebrate rigidity.

Read before the Section on Neuropsychiatry at the 77th Annual Session of the California Medical Association, San Francisco, April 11-14, 1948.

University of California Hospital, The Medical Center.

Heart Disease as a Complication of Pregnancy

With Emphasis on Indications for Recommending Therapeutic Abortion or Avoidance of Conception

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SUMMARY

Mere diagnosis of a valvular heart lesion without circulatory incapacity is inconsequential in considering the prognosis for a pregnant woman.

There are grave dangers of heart failure during pregnancy, labor or the postpartum period in women who have had congestive failure prior to pregnancy or during a previous pregnancy and delivery.

Decisions as to whether or not to recommend avoidance of pregnancy or therapeutic abortion should depend not alone upon the prospect of death during gestation or at the time of delivery, but also upon the possibility of increased impairment of heart function and physical incapacity of the mother in the postpartum period.

Because of the extensive surgical procedure there are few indications for interrupting pregnancy after the third lunar month; and because the major circulatory loads do not increase after the eighth month, rarely should labor be prematurely induced after that time.

The exertion of labor, although generally inconsequential, in some cases may produce sustained oxygen debt. In cases in which labor in a previous pregnancy has been accompanied by heart failure, cesarean section should be considered as a means of lessening the possibility of serious failure or death, although this is not a frequent consideration.

In the resemblance of circulatory changes that occur, during delivery and immediately postpartum, to those changes produced by the closure of an arteriovenous shunt or patent ductus arteriosus, lies a suggestion as to some of the causative factors in heart failure during or soon after delivery. Probably of great importance is the decrease in blood volume and hemoconcentration at delivery followed by the return of water to the circulatory system, with consequent transient increase in blood volume, in the postpartum period. Additionally, the rise of venous pressure after use of oxytocic drugs suggests that rapid infusion of blood from an engorged uterus may cause an abrupt and perhaps poorly tolerated hypervolemia.

Death by heart failure in pregnancy and the puerperium has become extremely rare in recent years due to the frequent observations, meticulous diagnosis of impending failure and improved care of cardiac patients jointly by the obstetrician and the internist. In addition to digitalis therapy and sodium restriction, patients exhibiting evidence of impending heart failure may require bed rest through the entire third trimester of pregnancy. Oxygen should be administered during labor in such patients and anoxia guarded against during anesthesia.

ALTHOUGH it has been reported that about 2.5 per cent of all women of childbearing age have heart disease, much of this proportion is made up of those who have mere anatomical cardiac defects which do not materially affect circulatory efficiency during pregnancy and delivery. Yet there remains a considerable number of women in this age field who have serious cardiac incapacities which might lead to grave heart failure or death under the additional stress of pregnancy and parturition, or to increased incapacity afterward.

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Presented as part of a Panel Discussion on the Complications of Pregnancy before a Joint Meeting of the Sections on General Medicine, General Practice, General Surgery, Obstetrics and Gynecology, and Public Health at the 77th Annual Meeting of the California Medical Association, San Francisco, April 11-14, 1948.

One of the purposes of this presentation is to consider the circumstances in which prevention or termination of pregnancy might be recommended with regard not only to the immediate hazard of death during gestation or delivery but also to the long-term effect on cardiac efficiency.

Until the current decade, obstetricians struggled in a welter of conflicting criteria for determining the degree of hazard of heart disease. Some believed aortic insufficiency to be the gravest lesion, others, including Lusk,[19] that mitral stenosis was so serious as to warrant termination of pregnancy. Through the time of Jensen's[16] publication in 1938 and Hamilton and Thomson's[14] in 1941, auricular fibrillation was considered to be associated with heart failure and fatality in about one-third of the cases. Mackenzie[20] stated in 1904 that a normal heart shows

derangement of function in pregnancy. All recognize that occasionally abrupt and critical failure with pulmonary edema occurs unexpectedly in certain previously well women late in pregnancy, during labor, or in the first 24 hours postpartum.

Within the past ten years, however, with information regarding larger series of individual studies of patients and with a better understanding of the circulatory loads in pregnancy, labor and the postpartum period, improved criteria have developed for use in deciding the advisability of proscribing conception or recommending early therapeutic abortion (within the first eight weeks) for women with heart disease.

The considerations upon which these criteria have been based are:

1. *Maternal mortality*,* which now approaches the vanishing point, possibly due to the application of the criteria outlined in the following paragraphs, as well as to the improved diagnosis and care of patients in cardiac-obstetrical clinics.

2. *Fetal mortality*—stillbirths and neonatal deaths. At New York University division of Bellevue Hospital[5] the fetal deaths at term were 9 per cent in mothers having cardiac disease without heart failure and 30 per cent in those with heart failure, as contrasted with 7 per cent in mothers without heart disease. There were no fetal deaths at term in the series at the University of California Hospital.

3. *Development of heart failure* with possibility of death—this to be considered not only as regards the immediate risk, but also as probably producing diminished circulatory efficiency in the future life of the mother.

4. *Effect on life expectancy.* Boyer and Nadas[1] found no difference in duration of life as between parous and nulliparous women who died of congestive heart failure. It is possible that such a statistical analysis is entirely correct, but it seems probable that this series of cases may not reflect occasional unfavorable effects of pregnancy upon women with heart disease.

5. *Impairment of circulatory efficiency.* As this has been found to be not unusual in patients with heart disease who have been subjected to physical effort of unusual degree or duration, it is logical to assume that the added physical loads of pregnancy and labor in certain instances may be of such degree as to have a similar deleterious effect. (This aspect will be discussed more fully in later paragraphs.)

From these basic considerations the following criteria have been evolved:

1. The mere anatomic existence of a particular valve lesion is not of prognostic importance; current or impending functional impairment alone is important.

2. *Both congenital and rheumatic lesions* are well or poorly tolerated in accordance with the mechanical handicap produced. Williams[28] and others have described a postpartal heart disease in which right and left heart failure occurs two to six weeks postpartum, with spontaneous recovery three to six weeks after onset. The cause of this phenomenon is unknown, but renal disease and hypertension are frequent concomitant findings.

3. *Auricular fibrillation* (as well as other arrhythmias, including complete A-V block) is not in itself a grave complication. Mendelson[23] of Cornell University Medical School reported no deaths in seven cases. However, because it is indicative of advanced cardiac disease, the greater likelihood of circulatory incompetence is a factor for consideration in such circumstances.

4. *Rheumatic activity* was reported by both Laubry and Routier[17] in France and Chavez[7] in Mexico as a grave and not uncommon complication. Hamilton and Thomson[14] fear recurrence when rheumatic fever has been active within six months of pregnancy. In women under 23 years of age, the chance of recurrence is 10 per cent greater than in older patients, as would be expected from Cohn and Lingg's[11] report that after seven years from onset, recurrence is unlikely. (The mean age at which the first attack occurs is 17 years.) Recurrence of rheumatic fever causes little concern in San Francisco; at University of California Hospital only five recurrences were observed among 90 pregnant women with rheumatic heart disease, and these were without influence on the courses of the patients during and after pregnancy.

*Unfortunately, reports of mortality rates offer the only standard for gauging from statistics originating in many quarters the deleterious effect of pregnancy and labor on women with heart disease. Lesser effects than death are not uniformly reported. By this one standard, however, considerable improvement has been recorded: There have been many reports of reduced mortality rates during pregnancy and labor among women with heart disease where cardiac-obstetrical clinics have been established. From 9.38 per cent prior to 1922, the rate fell to 3.24 per cent for the period 1936-46, according to a compilation by Bunim and Rubicus[5] of 23 reports covering an aggregate of 4,869 delivery cases. Even this rate is materially higher than that noted in reports from 1944 to 1947: Mendelson[23] stated that there were no deaths in 200 deliveries of patients with rheumatic heart disease, and Bunim and Rubicus[5] reported a mortality rate of 1.4 per cent, while Lund[18] reported one death in 29 deliveries of patients with congenital heart disease. In the 12 years 1936-48 there has been neither a maternal nor a term fetal death in the University of California Hospital in 119 patients with cardiac disease who were delivered there and who had been followed prior to delivery in the obstetrical-cardiac clinic. Although Hamilton and Thomson[14] stated in 1941 that there was an irreducible minimum death rate of 2.5 per cent due to subacute endocarditis, embolism, sepsis and hemorrhage, the use of antibiotics and anticoagulants has now reduced this "irreducible minimum."

The lower rate of deaths from heart failure where obstetrical-cardiac clinics have been established with trained internists or cardiologists in attendance is probably ascribable to (1) the more exact attention to the patients, (2) the greater diagnostic acumen of the internists in discerning the lesions and the early signs of heart failure, (3) the more expert use of drugs and physical management of heart failure, and (4) the gradual development, through experience, of the criteria for (a) permitting pregnancy, (b) abortion, (3) hysterotomy and (d) selecting the type of delivery—pelvic or cesarean—and the means of anesthetization.

5. *Age.* Hamilton found the incidence of heart failure in pregnant patients over 35 years of age to be double that in younger pregnant women. As is shown in Table 1, the incidence of lowered circulatory efficiency after delivery was thrice as great among women over the age of 35 as in younger women. Tendency toward heart failure in the older age groups has been attributed by Bunim and Rubicus[5] solely to the longer duration of the heart disease.

6. *Parity.* There is general conflict of opinion as to whether the likelihood of heart failure is greater in primiparous or in multiparous women with heart disease. Primiparas generally undergo greater physical and circulatory strain during labor, which could precipitate heart failure. Multiparas, on the other hand, grow older with each pregnancy and additionally often have more difficult labor after the fourth confinement. Eastman[12] reported a rise in mortality rate from 0.37 per cent in para 2 to 8 women to 1.15 per cent in those over para 8. Among para 8 women, one in five has hypertension or chronic nephritis. Longer duration of heart disease in women in the higher parity groups may explain other instances of congestive failure.

7. *Duration of heart disease* (rheumatic). The figures of Bunim and Rubicus show a rise in heart failure in pregnant women from 4 per cent in the first ten years after onset of rheumatic heart disease to 13 per cent between 10 and 14 years, to 18 per cent between 15 and 19 years and to 23 per cent 20 years and more after onset. (The progressive rise in incidence of failure coincides roughly with the increasing incidence of failure with advancing age.) This is an important criterion in considering the possibility of fatal outcome during or following pregnancy, since Cohn and Lingg[11] showed that the mean duration of life in all patients with rheumatic heart disease after onset of failure was three years. Using a more accurate statistical method, Wilson and Lubschez[4] present a more optimistic estimate of duration of life in patients with rheumatic heart disease.

Another factor for consideration in this regard is the relationship of the age of the patient to the severity of the heart disease. Hamilton[13] observed that whereas among women with heart disease of functional Classes I and II (favorable) the death rate between the ages of 20 and 40 years is the same for those who are pregnant as for those who are not; in Classes III and IV the annual death rate is 18 per cent for pregnant women against 6.7 per cent for the non-pregnant.

8. *Degree of failure before pregnancy* has long been recognized as a prognostic criterion. Among patients in Class III and IV, as found by Hamilton and Thomson[14] in their "unfavorable" group, the mortality rate from heart failure was 12 per cent, as contrasted with 0.5 per cent among patients in Classes I and II. Bunim and Rubicus reported that only one patient in a series of 82 in Classes I and II had serious failure in pregnancy, as contrasted with about one-third of the patients in Classes III and IV.

A high degree of failure, therefore, would seem to be the chief contraindication to pregnancy. However, as the statistics show a surprisingly high percentage of women in Classes III and IV who do not develop serious complications, the criterion is not absolute and there is margin for consideration of other factors in deciding upon advice and procedure. Even though the risk is appreciable, if the patient wishes to take it for personal reasons or because of religious beliefs, the percentages still favor successful outcome.

9. *Heart failure in a previous pregnancy*—unless transient and precipitated by some complication such as pneumonia, pulmonary embolism, active rheumatic carditis, paroxysmal auricular fibrillation or tachycardia or toxemia — is strong reason to expect recurrence of failure in a succeeding pregnancy (nine out of nine was the experience of Bunim and Rubicus). It is conceivable that, with greater rest, digitalis therapy and salt restriction, failure can be prevented, but this is certainly one of the strongest indications for preventing or interrupting pregnancy unless the risk is explained to and accepted by the patient.

10. *The influence of hypertension* on the outcome of pregnancy has not yet been ascertained, but this disease probably does not produce a serious complication. Sharkey and Hess[25] reported that there were neither fetal nor maternal deaths in a series of 118 pregnant women with essential vascular hypertension. Chesley, Annito and Jarvis,[8] from observation of 301 pregnancies in 218 women with hypertension, concluded that the condition was hazardous only insofar as it affected the chances of occurrence of toxemia. However, the incidence of toxemia in hypertensive patients was seven times the normal rate of occurrence, with maternal mortality 20 times normal and fetal mortality ten times normal. Apparently, then, previous history of toxemia in association with hypertension would be a relative contraindication to pregnancy.

DECREASED CIRCULATORY EFFICIENCY

As to the previously mentioned question of increased impairment of circulatory efficiency by reason of pregnancy, an additional consideration is the future well-being of the patient and the effect upon her household. If pregnancy and parturition are likely to result in greatly diminished capacity of a woman to take care of her children and household, leading to a social and economic problem, this is an added contraindication to the continuance of pregnancy when such dangers are predictable by the criteria outlined. To assume that maternal death or complete recovery are the only extreme consequences of pregnancy and labor in women with heart disease, and that no intermediate results of increased crippling should be considered, seems illogical, although it is a widely accepted attitude.

Present data from the University of California obstetrical-cardiac clinic (Table 1) show that approximately 12 per cent of the patients under 25 years of age and 33 per cent of patients above that

TABLE 1.—*Influence of Pregnancy on Postpartum Circulatory Efficiency, Relative to Age, Parity, Mode of Delivery (107 Patients).*

Parity	Mode of Delivery	No. of Patients	Dim. Postpartum Funct.	Percentage
	UNDER AGE 35			
Grav. I	Pelvic	35	5	14.3
	Cesarean	3	0	.....
	Abort. or Hyst.	1	0	.....
Total		39	5	13.0
Grav. II-IV	Pelvic	37	5	13.5
	Cesarean	4	0	.....
	Abort. or Hyst.	2	0	.....
Total		43	5	11.6
Grav. V-IX	Pelvic	11	1	9.1
	Cesarean	1	0	.....
	Abort. or Hyst.	1	0	.....
Total		13	1	7.6
TOTALS	PELVIC	83	11	13.2
	CESAREAN	8	0	.....
	ABORT. OR HYST.	4	0	.....
		95	11	11.6
	OVER AGE 35			
Grav. I	Pelvic	0	0	.....
	Cesarean	0	0	.....
	Abort. or Hyst.	0	0	.....
Total		0	0	.....
Grav. II-IV	Pelvic	6	1	16.6
	Cesarean	1	0	.....
	Abort. or Hyst.	0	0	.....
Total		7	1	14.3
Grav. V-IX	Pelvic	5	3	60.0
	Cesarean	0	0	.....
	Abort. or Hyst.	0	0	.....
Total		5	3	60.0
TOTALS	PELVIC	11	4	36.4
	CESAREAN	1	0	.....
	ABORT. OR HYST.	0	0	.....
		12	4	33.3
	COMBINED TOTALS			
Grav. I	Pelvic	35	5	14.3
	Cesarean	3	0	.....
	Abort. or Hyst.	1	0	.....
Total		39	5	12.8
Grav. II-IV	Pelvic	43	6	14.0
	Cesarean	5	0	.....
	Abort. or Hyst.	2	0	.....
Total		50	6	12.0
Grav. V-IX	Pelvic	16	4	25.0
	Cesarean	1	0	.....
	Abort. or Hyst.	1	0	.....
Total		18	4	22.0
TOTALS	PELVIC	94	15	16.0
	CESAREAN	9	0	.....
	ABORT. OR HYST.	4	0	.....
		107	15	14.0

Of those showing postpartum diminished cardiac function, 6 out of 15 were Class III or IV.

Of those not showing postpartum diminished cardiac function, 11 out of 92 were Class III.

age have diminished ability to undertake physical work after delivery. That multiparity may have some further effect in this connection is indicated by the fact that three out of five patients over the age of 35 who were para 5 to 9 experienced a reduction in physical capacity. No attempt was made in these cases to demonstrate frank failure by measurement of venous pressure or circulation rate, inasmuch as most of the patients did not show objective signs of heart failure and only 20 per cent had functional impairment of an order greater than Class II. It is recognized, therefore, that psychic factors rather than true dynamic circulatory insufficiency may have caused some of the diminished physical capacity in these patients. In this regard it is noteworthy that approximately two-thirds (11 out of 17) patients with Class III and IV impairment were apparently as physically capable after pregnancy as before. However, that reduction in circulatory efficiency occasionally does occur even in Class I and II patients should be evident to all clinicians making follow-up postpartum observations of patients with heart disease.

A case report will serve to illustrate this point as well as the application of the criteria for determining whether to terminate pregnancy in an early stage or permit it to continue.

CASE REPORT

The patient, aged 24, when examined at the obstetrical-cardiac clinic in the ninth week of pregnancy gave a history of acute rheumatic fever at the age of 11 years which involved the extremities, jaws and spine. Attacks lasting a few days recurred about every six months. The last attack had been three months previously, when the patient spent a week in bed, with fever and with pain and swelling in the knees, ankles and elbows.

It was not until the patient was 19 years of age that a valvular defect had been diagnosed, but dyspnea had been present on running or climbing since the original attack of rheumatic fever. During a first pregnancy, a year and a half before the present one, the patient had felt well until dyspnea and orthopnea recurred in the sixth month. Digitalis was given during the remainder of that pregnancy and the patient felt improved in the ninth month although limited to chair and bed.

After delivery, the patient stopped taking digitalis and she then noticed more dyspnea than in the last months of pregnancy, especially when climbing stairs. She felt that the dyspnea was definitely worse then than it had been prior to pregnancy.

Upon physical examination the blood pressure was 124 mm. of mercury systolic and 74 mm. diastolic, and the pulse rate was 75 at rest, 112 following exercise. There was no edema and no rales were noted. The chest was slightly funnel-shaped. A roentgenogram revealed the heart to be moderately enlarged, but an electrocardiogram tracing showed no abnormality. Vital capacity was 1,600 cc. Erythrocyte and leukocyte counts and the blood sedimentation rate were within normal limits. There were characteristic signs of mitral stenosis and insufficiency and aortic insufficiency.

In this case the results of applying the previously outlined criteria were as follows:

Indications for termination of pregnancy:

1. Definitely increased dyspnea during sixth to ninth month of previous pregnancy. Functional Class III in second month of current pregnancy.
2. Recurrence of rheumatic fever within six months of current pregnancy.
3. Dyspnea worse after pregnancy than prior to or even during eighth or ninth month of pregnancy (when activity was limited).
4. Questionable ability to care for two children.
5. Thirteen years since onset of rheumatic heart disease.
6. Danger of serious heart failure with any intercurrent complications, such as respiratory tract infection.

Reasons for continuation of pregnancy:

1. Strong desire for second child.
2. Assurance of household care during pregnancy and of assistance afterward.
3. Available hospital and medical care during pregnancy.
4. Multiparity, with previous delivery by low forceps after two hours of "easy" second stage labor.
5. No current rheumatic fever.
6. Religious belief relative to abortion.
7. Current conservative attitude of obstetrical hospital staff.

The pregnancy was not terminated and heart failure did not develop. Indeed, after delivery the patient showed less evidence of insufficiency than when first observed early in pregnancy, possibly because of enforced rest in the last trimester.

CRITERIA FOR LATER TERMINATION OF PREGNANCY

The criteria for interruption of pregnancy after two and a half months are still more unsettled than those for prevention or early abortion. In years past, hysterotomy was not uncommonly done between the sixth and ninth month of pregnancy in the presence of persistent heart failure. Hamilton[14] reported hysterotomies in 7.5 per cent of such cases up to 1941, but has recommended the operation rarely since 1944. No hysterotomies were performed by the New York University group,[5] and whereas four were done at the University of California Hospital in the first five years of the obstetrical-cardiac clinic, there have been none in the past seven years.

Other than the development of means for meticulous care of patients with heart failure by bed rest and the administration of digitalis and diuretics, the chief reason for this change of attitude has been an appreciation of the nature and timing of the circulating loads of pregnancy.

An increase in load begins to appear about the fifth lunar month, and according to Cohen and Thomson[10] (Figure 1) and Burwell[6] there is continued increase until midway of the ninth lunar month, when the load diminishes about 10 per cent from the peak. (This late decrease is not confirmed by McLennon and Thoun.[21])

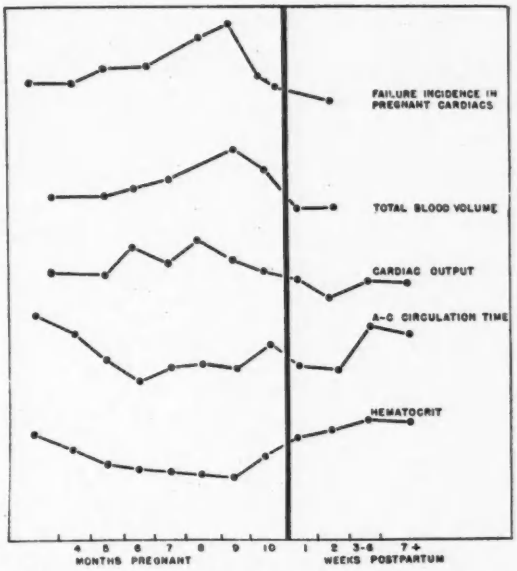

Figure 1.—Circulatory burden on the heart in pregnancy (from Cohen and Thomson, J.A.M.A., 1939, 112:116).

Explanation of the rising loads lies in:

1. Increase of total blood volume by an average of 32 per cent (McLennon and Thoun[21]) and a maximum of 45 per cent. As the plasma volume increases to a greater extent than the cell mass, in a ratio of about 2:1, a false appearance of anemia results because of the relatively smaller content of packed red cells in the increased volume of whole blood.

2. Increase in cardiac output to a maximum of 50 per cent.

3. Increase in basal metabolic rate to, generally, a maximum of 12 per cent.

4. Increase of body weight from three sources, totaling an average of 6.67 kg.: (a) The products of conception, rarely over 5 kg. (1,500-2,000 cc. of water in fetus, placenta and amniotic fluid)[9]; (b) gain in maternal fat tissue, an unnecessary load controllable by diet; (c) water gain (maternal) averaging 2.5 kg. or about 2,500 cc. About one-fifth of all obstetrical patients show abnormal water gain, and in them there is a tendency toward preeclamptic states and edema. About 60 per cent of this maternal water is lost in the first six days postpartum.

Cohen and Thomson[10] have observed that the incidence of heart failure increases through the eighth lunar month of pregnancy and then diminishes to term. This has not been confirmed by Mendelson[23] or Bunim and Rubicus[5] who found the onset of failure equally distributed from the fifth to the tenth lunar month. It is important to recognize evidence of impending heart failure if proper treatment is to be instituted. Certain changes have been found to occur in the circulatory system during pregnancy in normal women, and deviations from these normal changes should alert the physi-

cian to the possibility of heart failure. These normal changes are:

1. Increase in vital capacity by about 10 to 15 per cent maximal to term. The measurement of vital capacity is a very important routine clinical test.

2. Venous pressure[22] in the upper extremities increases only 1 to 2 cm. of water after the 25th week of pregnancy. In the lower extremities it increases in the last 30 weeks of pregnancy.

3. Circulation time generally diminishes from the 20th to the 30th week.

4. Blood pressure. Normally there is elevated pulse pressure through the last four months of pregnancy. Commonly there is a slight fall in systolic and diastolic pressure in the middle trimester, with recovery in the last trimester—not to be considered evidence of impending toxemia.

5. Diminished arteriovenous oxygen difference. This is not commonly measured clinically.

6. Increased blood volume with hemodilution as evidenced by hematocrit measurement.

Why these changes in the blood volume, in the metabolic rate and in the arteriovenous oxygen difference occur is not entirely explained.

The theory of Burwell[6] that the placenta acts as an arteriovenous shunt may well be the answer to most of these changes. Probably the variation in estrogenic hormone content causes a negative sodium balance. Thorn and Emerson[27] showed sodium and water retention in cycles at the premenstrual period in hysterectomized patients as well as in normal women, and with estrogen therapy in castrated women. Taylor, Warner and Welsh[26] reported preventing postpartum diuresis with estrogen administration. The similar action of desoxycorticosterone acetate is well known and, hypothetically, the pituitary antidiuretic hormone may play a role in water retention and possibly in hypervolemia.

Additionally it is known that there is increase in the volume of the peripheral vascular bed in patients with abnormally high metabolic rates, as in hyperthyroidism. This increase in the peripheral vascular bed is also associated with a gain in blood volume and as such may be a partial explanation of the hypervolemia of pregnancy.

TYPE OF DELIVERY

The determination of type of delivery in the presence of heart disease has been a subject of much discussion. Data reported by Hoffman and Jeffers[15] indicate that, among patients with rheumatic heart disease, deaths in the first two postpartum days occur as commonly following delivery by cesarean section as by the pelvic route. Reports by Hamilton[13] and Mendelson[23] indicate a mortality rate of 12 per cent after cesarean section as against no deaths after pelvic deliveries in Class III and IV patients. Bunim and Rubicus have expressed the opinion that heart disease is never an indication for cesarean section. That these statistics ought to be weighed in considering the interruption of pregnancy or cesarean section in critical cases is evident, but no doubt exists that important circulatory changes occur during delivery and the postpartum period irrespective of the type of delivery.

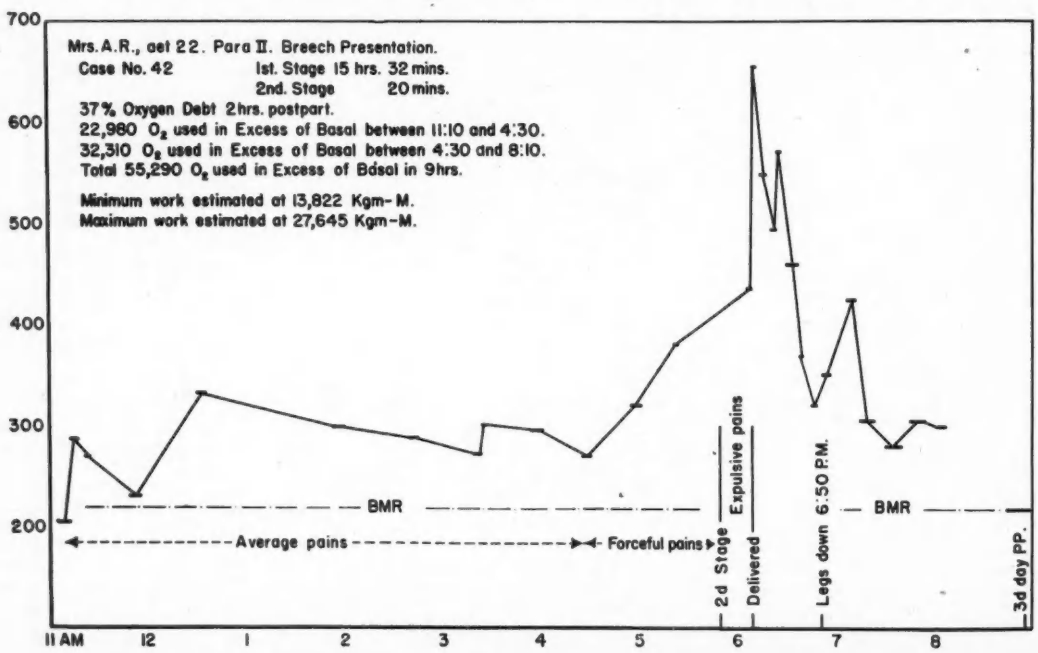

Figure 2.—Rate of oxygen consumption during and following labor as evidence of physical work of labor (Sampson et al.[24]).

To evaluate the mechanical effect of labor in producing circulatory strain, Sampson, Rose and Quinn[24] estimated the physical work of labor and the residual "oxygen debt" in 40 women by periodic determinations of oxygen intake during and following labor. The conclusions of this study were (1) that the work may be negligible, but (2) that even in multiparous women, if a complication develops, there may be abrupt and considerable loads of work undertaken, equal to climbing a 30-story building in about one hour (Figure 2); (3) that "oxygen debt" persisted for more than an hour in patients with heart failure and also in normal women, chiefly primiparous who apparently had performed heavy work; (4) that work of this degree would be proscribed for non-pregnant patients of Class III and IV on the grounds of experience that failure may be precipitated by heavy work; (5) that cesarean section is well tolerated on the operating table and that the dangers surgically and from postpartum heart failure must be weighed against the benefits of relief from expenditure of an indeterminate amount of physical energy in labor. Occasionally a history of previous precipitation of heart failure by labor would indicate cesarean section to avoid failure in a subsequent pregnancy.

Circulatory changes other than those caused by oxygen uptake and transport have been measured by Brown, Sampson, Wheeler and Giansiracusa[2] during and immediately following labor. Included were measurement of blood volume, packed cell volume, venous pressure, circulation rate, pulse rate and blood pressure (Figure 3). The conclusions from this study were:

1. That, except in frank heart failure, rise in venous pressure occurred only following use of oxytocic drugs. This may be explained by the sudden infusion of semi-pooled blood from an engorged uterus into the systemic circulation. Abrupt delivery of blood from the uterus, as at cesarean section, may be less well tolerated than the slower emptying by the repeated uterine contraction of labor.

2. That changes in pulse rate and blood pressure immediately after delivery suggest in a significant percentage of cases the same process as that which occurs with the obliteration of an arteriovenous shunt.

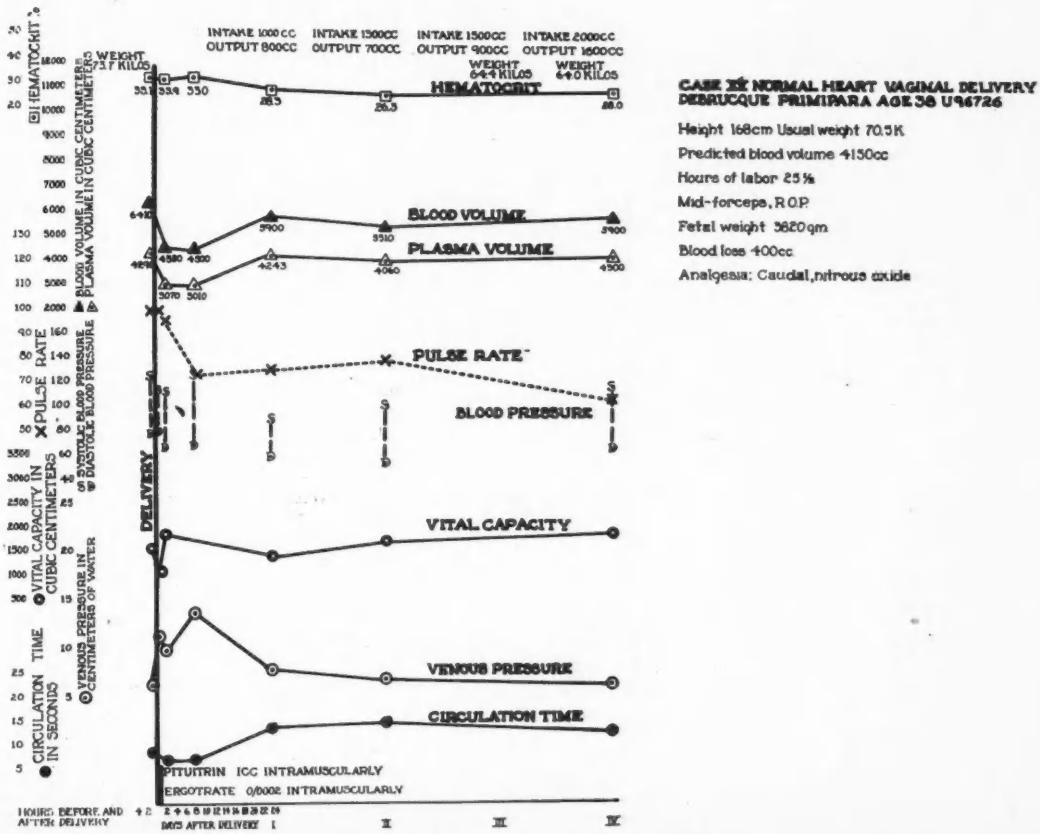

Figure 3.—Measurement of certain circulatory functions in a normal patient immediately prior to and following normal delivery (Brown et al.[2]).

3. That a fall of blood volume and hemoconcentration occurs after delivery but that there is a partial reversal of this decline before normal stabilization in three to five days. Such a process also occurs after ligation of patent ductus arteriosus, according to Brown,[3] and in some such cases heart failure seems to accompany the return of the blood volume. This is one of the first clues, although still to be confirmed, as to a possible cause of postpartum cardiac failure or death.

Finally, studies made by Brown, Hopper, Mudrick and Sampson[4] showed that repeated periods of Valsalva's maneuver (forced expiration with closed glottis) with elevated venous pressures can cause significant filtration of water out of the blood stream. As the Valsalva effort duplicates in some degree the mechanics of labor, the studies may indicate why hemoconcentration follows delivery.

THERAPY

During pregnancy, bed rest, digitalis, diuretics and restriction of sodium intake may be used in combatting heart failure, quinidine in prevention or correction of arrhythmias, and antibiotics in treatment of infections and recurrence of rheumatic fever. Oxygen inhalation may be necessary at times; it is particularly important during labor. Great care should be taken to avoid anoxia during anesthesia. For this reason the use of nitrous oxide and oxygen to obtain deep anesthesia is considered inadvisable since anoxia almost invariably accompanies such anesthesia.

Suggestions for therapy to be applied to patients with heart failure during the immediate postpartum period are (1) restriction of oxytocic drugs as much as possible because of their precipitation of an abrupt rise in venous pressure, (2) mercurial diuresis, (3) possibly withdrawal of blood, unless anemia is present, and (4) a longer period of postpartum bed rest than is given to the normal patient.

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Surgical Treatment of Patent Ductus Arteriosus

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SUMMARY

Since it would appear that diagnosis of patent ductus arteriosus can be made accurately in a high percentage of cases, and as the surgical treatment of this lesion is highly successful with a low mortality and morbidity rate, operation is advisable for all patients with this lesion except those who are 35 years of age or older and who have no progressive cardiac hypertrophy and no incapacitation due to the fistula. The optimum age for operation is three years. The vast majority of the patients have a ductus that can be divided, and division has advantages over ligation.

In cases in which patent ductus arteriosus is complicated by subacute bacterial endocarditis, operation should be done following intensive penicillin therapy.

TEN years ago Gross[1] first successfully ligated a patent ductus arteriosus and since then physicians have shown a greater interest in the accurate diagnosis of congenital cardiac lesions and surgeons have met the challenge, either to alleviate or to cure these malformations. Great advances have been made in the production of fistula shunts in patients having pulmonary artery stenosis and in the resection of coarctations with end-to-end anastomosis of the aorta. More recently Smithy resected the scar of a mitral stenosis with a valvulotome introduced through the ventricle, and the result was great clinical improvement of the patient. Experimental surgery gives promise of successful closure of interauricular septal defects, although the author knows of no case in which it has been attempted in a human. These newer and more recent spectacular operations have overshadowed the surgical ligation and division of the patent ductus arteriosus which have now become commonplace surgical procedures that no longer create the comment or the attraction that they invited so recently as three years ago.

SYMPTOMS AND PHYSICAL FINDINGS

In patent ductus arteriosus there may be no symptoms whatsoever, and the patient may appear well and normal. Palpitation, weakness, ease of fatigue and inability to keep up with the activities of other children are not uncommon. Tachycardia, fainting attacks, dyspnea on exertion and epistaxis are occasionally complained of, and, finally, the usual symptoms of cardiac failure and of subacute bacterial endocarditis may be noted in the more acutely ill patients with complications.

Although most of the patients are slim and underdeveloped, some appear normally developed in all respects. Retarded physical growth and development are attributed by Eppinger and Burwell[1] to the shunt of blood from the aorta to the pulmonary artery with a diminution of the peripheral flow. These patients have no clubbing of the digits or cyanosis unless there exists another congenital lesion for which the patent ductus arteriosus may be an accommodating fistula or unless the patient is in heart failure.

An active precordial heave and visible pulsation of the cervical vessels may or may not be noted, but a palpable thrill is felt over the pulmonic area in the vast majority of patients. The thrill may be systolic or continuous with systolic accentuation and may not appear until the third or fourth year of life. In some of the patients observed by the author, detection of the thrill by the mother led to the patient's being examined and the diagnosis is made. If the thrill is absent in a patient over four years of age who has patent ductus arteriosus one might speculate that the ductus is small.

The murmur may be absent or easily missed in infants because of the rapidity of the heart and the small size of the chest, but usually after the third year the murmur is definitely a characteristic one; it is a continuous rumbling "machinery murmur," usually accentuated during systole or early diastole, maximum in intensity in the pulmonic region, and transmitted over the precordium to the back and to the axilla. In all cases in which a continuous murmur was questioned clinically, an electrical sound recording of the murmur proved it to be continuous. The second pulmonic sound was increased or reduplicated in almost all cases observed by the author.

The vast majority of patients observed had elevated pulse pressure with low diastolic blood pressure, and the latter usually was difficult to measure accurately, there being no sharp point which could be read with certainty. Exercise, exertion and excitement caused pronounced increase in the pulse pressure and lowering of the diastolic pressure in most patients. Corrigan pulse and pistol-shot sounds over the femoral arteries may be observed in patients with a large ductus. Blood pressure readings are taken in both upper and lower extremities to detect coarctation of the aorta.

Usually, although not in all cases, the heart is

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slightly enlarged. Ordinarily the heart rate is increased and the intensity and vigor of the heart action is greater than normal; exercise definitely increases the rate and the intensity of the heart beat more than would be expected in a normal patient.

LABORATORY FINDINGS

Electrocardiographic studies usually show no abnormality and no axis deviation because the left ventricle pumps a larger volume per minute and the right ventricle must pump against an elevated pressure in the pulmonary arteries, the additional loads more or less counterbalancing one another. Axis deviation should arouse suspicion of the presence of another lesion, and a right axis deviation would particularly suggest pulmonary stenosis.

Fluoroscopic and roentgen studies usually demonstrate some cardiac enlargement, prominence of the pulmonary artery, increased pulsation of the heart and great vessels and pulmonary congestion. These abnormalities, however, are not in themselves specifically diagnostic findings of patent ductus arteriosus; they are merely confirmatory evidence.

INDICATIONS FOR OPERATION

In formulating an opinion as to the advisability of operation in uncomplicated patent ductus arteriosus, the chances of development of complications in the untreated patient must be weighed against the dangers of operation. The author's experiences in 100 cases treated surgically warrant advising operation in all patients with this lesion except those in their thirties or older who have no progressive cardiac hypertrophy or incapacitation due to having had a shunt for so long. The optimum age appears to be about three years, although the author has operated upon some patients younger than that. The older the patient the more difficult the operation technically. The vessels of the young patient are far more elastic and the tissues may be dissected with ease, in contradistinction to those of the adolescents and adults. If patent ductus arteriosus is complicated by subacute bacterial endocarditis, operation should be done after prolonged and intensive treatment with penicillin. The fact that penicillin may cure bacterial endocarditis does not justify advising a patient not to have the operation. It must be assumed that the damage done by infection is not eradicated by penicillin. Moreover, an apparent cure by penicillin does not necessarily prevent recurrence of subacute bacterial endocarditis. The fact remains that the presence of a shunt throws a burden on the heart so that in the majority of cases cardiac hypertrophy develops. Furthermore, the newer technique of dividing the ductus and suturing the wall of the pulmonary artery and aorta, not only prevents the chance of reestablishing the shunt but it also offers greater assurance against the development of subacute bacterial endocarditis. In young patients there is no sure method of predicting in which ones cardiac hypertrophy or subacute bacterial endocarditis will develop; and as long as the surgical morbidity and mortality rate is very low, it is most urgent to treat these patients surgically.

OPERATIVE TECHNIQUE

The author uses a posterolateral transpleural approach, resecting a long segment of the fourth rib posteriorly. With the rib spreaders applied, the operative exposure is far superior to that obtained by the anterior approach which the author used in the first 41 cases. The lung is allowed to collapse, the mediastinal pleura over the ductus is incised and the ductus is dissected all the way around. Cotton tapes are passed around the ductus to aid in a thorough and complete dissection of the tissues about the vessel and the reflection of tissue from the pericardium over the ductus, pulmonary artery and aorta. Four hemostats are placed across the ductus, which is then divided between the inner two hemostats as proposed and described by Gross.[3] Then the inner two hemostats are removed and both cuffs of the ductus distal to the remaining hemostats are sutured with fine silk on atraumatic needles. The remaining two hemostats are then removed and the suture line covered with pledgets of gelfoam. The mediastinal pleura is then sutured and after the lung is expanded the chest wall is closed in layers without drainage.

It is the author's belief that division of the ductus rather than ligation in continuity should be done in every instance in which it is surgically feasible. Occasionally the ductus may not appear long enough, but if the dissection is thorough, in the vast majority of such cases it can be "lengthened"; and the clamps may encroach a trifle on the walls of the vessels to permit the division of the ductus. Mobilizing the aorta and pulmonary artery is a great aid in preparing the ductus for division, and the experienced surgeon will have no difficulty getting enough length of the ductus to divide. The division of the ductus insures against reestablishment of the fistula and possible subsequent development of subacute bacterial endocarditis and is therefore preferable to the procedure of simple ligation.

Ether-oxygen endotracheally has been used for anesthesia in the vast majority of these operations and is preferred to Cyclopropane (trimethylene).

POSTOPERATIVE CARE

Postoperative care has been rather simple. Very few of the patients have required glucose infusions; blood transfusion is contraindicated except where a hemorrhage indicates its use; and in the last 30 operations only four patients have required pleural aspirations. Penicillin is now administered for 24 hours before operation and for 72 hours postoperatively. Usually the patients are discharged from the hospital seven days after operation.

OPERATIVE COMPLICATIONS

Recently a detailed account of the postoperative complications in the first 61 patients of this series

was reported.[4] In the last 39 cases, only five patients have needed pleural aspirations and only one has had a disturbing complication—namely, transient pericardial effusion which absorbed spontaneously and which kept the patient in the hospital only 16 days instead of the usual seven days postoperatively. Convalescence in this case was uneventful. Briefly, the complications in 100 successive operations (58 ligations and 42 divisions) were as follows: 27 patients required pleural aspiration; two developed empyema necessitating rib resection (both made good recovery); in two of the earlier cases in which ligation was done with umbilical tapes, aortobronchial fistula developed at the site of the tied knots. In one of these, surgical intervention was necessary, while in the other healing was spontaneous. Both patients have been well for a long time. These cases were reported in detail elsewhere.[4] Five patients had pulmonary atelectasis which cleared with more intensive administration of carbon dioxide and postural treatments. In two cases in which ligation had been done, persistent recurrent laryngeal nerve palsy developed although meticulous care had been taken to avoid injury at the time of operation. These cases were among the earlier in the series, and it is assumed that the irritation or pressure of the knots of tape in close proximity to the recurrent laryngeal nerve caused this complication. There were no wound infections but the incidence of keloid in the supramammary scars was one of the reasons for abandoning the anterior approach.

Two patients in the series of 100 died. One, a child operated upon in 1939, developed Staphylococcus aureus endarteritis about 30 days after ligation of the ductus and died 48 days postoperatively. The ligatures had cut through the walls and a superimposed blood stream infection resulted in vegetations on both pulmonary artery and aortic walls. The details of this case are reported elsewhere.[5] The other died of cardiac arrest following hemorrhage during dissection of the ductus.[4] In the last 40 cases (35 divisions and five ligations) no serious complication has occurred.

RESULTS

The beneficial effects of the operation in children with developmental retardations has been very obvious. Gain in weight, appetite and strength and capacity for activity has been a universal effect. Patients who had palpitation, tachycardia, fainting spells and ease of fatigue were soon completely asymptomatic. A rise in diastolic blood pressure and a decrease of the pulse pressure were noted either at once or within the first few days after operation. A decrease in the size of the heart in all patients who had cardiac hypertrophy before operation was noted within a month or two afterward.

In all of the 100 cases, patent ductus arteriosus was found at operation and either ligated or divided; there were no mistaken diagnoses. Ninety-eight of the 100 patients operated upon are alive, and only one of them is not entirely well. In that one patient, a diabetic, a fistula has reestablished itself since ligation several years ago, and the patient has a low cardiac reserve. None of the 42 patients who were operated upon for division of ductus had consequential residual murmur, but ten of the 58 in whom ligation was done had a continuous murmur which persisted or which returned later after discharge from the hospital. The importance of the surgical division of the ductus, rather than ligation, is borne out in this series.

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Substernal Goiter and Pulmonary Emphysema

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SUMMARY

Pulmonary emphysema is not an infrequent complication of tracheal compression due to goiter.

In many cases the goiter is in the mediastinum and therefore overlooked.

Among 13 patients with this combination of disease, chronic cough, dyspnea and wheezing were the chief respiratory symptoms. Ten of the 13 patients also had hyperthyroidism; in eight of the ten the disease was of the "masked" variety and both the thyrotoxicosis and the mediastinal mass were overlooked.

Early recognition and surgical removal of substernal goiter may prevent or retard otherwise irreversible and progressive tracheobronchopulmonary disease.

Medical management of such cases with thiouracil preparations may lessen the toxic symptoms but probably will not affect the mechanical compression of the trachea.

THE symptoms of tracheal compression due to substernal or intrathoracic goiter have been adequately described by many authors.[2, 3, 8, 9] Cough, paroxysmal or constant dyspnea, wheezing, asthmatic attacks, "choking spells," recurrent bronchitis and hoarseness are the principal symptoms but all are not necessarily present in a given case. Such symptoms may readily be ascribed to primary bronchopulmonary disease and the goiter may be overlooked. This is especially true when there is no goiter in the neck. Dramatic relief of the symptoms of obstruction frequently follows operation, but irreversible tracheal and pulmonary changes may persist if the tracheal obstruction has been of long duration.[2]

For some time the authors have been impressed with the high incidence of chronic pulmonary emphysema in patients with substernal goiter. These patients had long-standing tracheal obstruction due to substernal goiter which was not diagnosed. For the most part they had "masked" hyperthyroidism[4, 5, 6, 7] without significant enlargement of the thyroid gland in the neck. Furthermore, the substernal goiter was not always apparent in the routine posterior-anterior chest film because the gland was small or localized under the manubrium (Cases 10 and 13 in Table 1). Fluoroscopy and lateral and oblique x-ray views were sometimes necessary to visualize the mediastinal mass and the tracheal compression.

It is pertinent at this time, when thyrotoxicosis is often treated with antithyroid substances without operation, to reemphasize the purely mechanical complications of goiter. The possibility of tracheal compression should be considered in all patients with goiter or hyperthyroidism, especially when there are symptoms or signs of bronchopulmonary disease. It might even be wise to adopt the attitude that substernal goiter should be considered in all cases of emphysema.

Clinical data of 13 selected cases which illustrate different aspects of this problem are summarized in Table 1. In every instance there was x-ray evidence of pressure on or distortion of the trachea. All but three patients (Cases 2, 5, and 8) had hyperthyroidism. In no instance was there a history of bronchial asthma; personal and family histories of allergic manifestations were not present, nor was there eosinophilia. Factors predisposing to chronic bronchopulmonary infection, such as pertussis or other acute or recurrent infections, were not found. There was no evidence of primary pulmonary disease such as tuberculosis or silicosis.

Cough was the most common symptom (nine cases) and was present for at least one year before treatment. If the cough had been present for two years or less, considerable improvement followed removal of the obstructing goiter (Cases 7, 8, and 12). The degree of improvement in the other respiratory symptoms such as difficulty in breathing, hoarseness, "asthma," wheezing, or strangling when recumbent also largely depended upon the duration of tracheal obstruction. In some cases the thyrotoxicosis *per se* accentuated the degree of dyspnea. The only complete cures of respiratory symptoms occurred in a 28-year-old woman who had had symptoms for only 18 months (Case 8) and in a 68-year-old man who had had hoarseness and dysphagia for only three months (Case 2). On the other hand, a 52-year-old male who had no respiratory symptoms but in whom there was objective evidence of obstructive emphysema developed progressive pulmonary changes during the next 17 years even though the tracheal compression was relieved (Case 4). He is now 70 years old and well except for emphysema. In this case the pulmonary changes had become irreversible before subjective symptoms appeared. It is not surprising that the patients who had had respiratory symptoms for five to twenty years showed no improvement after operation (Cases 1, 6, 11, and 13). Case 11 warrants a more complete report because it illustrates the difficulties

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in diagnosis and the complications of prolonged tracheal obstruction and chronic pulmonary emphysema.

CASE REPORT

CASE 11.—A 52-year-old male was admitted to the Peter Bent Brigham Hospital, Boston, on February 14, 1945, complaining of paroxysmal nocturnal difficulty in breathing which was relieved by sitting upright in bed. This difficulty had been present for 20 years. For at least 12 years the patient had been exceptionally nervous and noted frequent "heaviness" over the sternum with excitement or exertion, and for ten years he had had an almost continuous hacking cough and dyspnea even at rest. According to the patient, medical consultants had ascribed these symptoms to a "chronic nervous breakdown." During the preceding year the patient had noted irregular pulse, orthopnea and ankle edema. Direct questioning revealed that he had

TABLE 1.—*Clinical Data on 13 Patients with Substernal Goiter*

Case	Symptoms	Goiter	Trachea by X-ray	Chest Findings	B.M.R.	Postoperative Result
1.—Female Age 63	Cough, dyspnea, 5 yr. Weakness, 6 wk.	205 gm. thyroid mainly substernal	Narrowed	A-P diameter increased. Wheezes.	+57%	Cough and wheezes present 2½ yr. later although trachea normal. Thyrotoxicosis cured.
2.—Male Age 68	Dysphagia, hoarseness, 3 mo.	Entirely intrathoracic; behind trachea and esophagus	Compressed and displaced to right	A-P diameter increased. Hyperresonant. Breath sounds diminished.	Not done	Immediate relief of symptoms.
3.—Male Age 61	Dyspnea, 2 yr. Life-long cough.	Entirely intrathoracic (toxic adenoma)	Compressed and displaced to left	A-P diameter increased. Breath sounds diminished. Rales in bases.	+27%	Trachea normal. Cough and respiration improved. Died 3½ yr. later of heart failure.
4.—Male Age 52	Nervous, palpitation, weight loss, 10 wk.	Slightly enlarged in neck. Gland mostly substernal	Narrowed	A-P diameter increased. Hyperresonant. Coarse rales.	+98%	Progressive emphysema and bronchitis during next 17 yr. although tracheal obstruction removed.
5.—Female Age 52	Strangling, wheezing, 1 yr. Swollen neck, 10 yr.	Large nodular goiter in neck extended below manubrium	Narrowed and displaced to right	Wheezes.	+10%	Trachea normal. Respiratory symptoms unchanged 1½ yr. later.
6.—Male Age 45	Cough, 10 yr. Dyspnea, perspiration, 1 mo.	Mostly substernal; nodule in right lobe	Narrowed and displaced to left	A-P diameter increased. Hyperresonant. Breath sounds diminished. Coarse rales.	+75%	Trachea normal. Thyrotoxicosis cured. Cough still present 2½ yr. later.
7.—Male Age 63	Dyspnea, cough, 1 yr.	Mostly substernal; small nodule in right lobe	Narrowed and displaced to right	A-P diameter increased. Breath sounds diminished.	+74%	Dyspnea gone. Slight cough persisted 3 mo. later.
8.—Female Age 28	Cough, dyspnea, 1½ yr.	5x6 cm. nodule in right lobe with substernal extension	Narrowed and displaced to left	Expiration prolonged.	—6%	Complete relief 1 mo. later.
9.—Female Age 65	Cough, 40 yr. Weakness, anorexia, 5 yr.	Mostly substernal	Compressed anteriorly	A-P diameter increased. Breath sound diminished. Many basal rales.	+41%	Thyrotoxicosis cured. Moderate cough persists 10 mo. later.
10.—Male Age 55	Angina, 3 yr. Tremor, 2 yr. Heat intolerance, 1 yr.	Entirely intrathoracic	Compressed anteriorly	Barrel-shaped. Breath sound diminished. Wheezes.	+36%	Under treatment with propyl thiouracil (see text).
11.—Male Age 56	Nocturnal dyspnea, 20 yr. Cough, 10 yr. Nervous, perspiration, 12 yr.	Almost entirely intrathoracic	Narrowed and displaced to left	Barrel-shaped. Hyperresonant. Breath sound diminished. Wheezes.	+30%	Progressive emphysema with wheezing, cough and development of cor pulmonale during next 3 yr.
12.—Female Age 42	"Asthma," choking spells, cough, 2 yr.	250 gm. thyroid almost entirely intrathoracic	Narrowed and displaced to right	A-P diameter increased. Wheezes.	+23%	Greatly improved. Slight cough and wheezing only with colds during next 8 yr.
13.—Male Age 57	Asthma, cough, 5 yr. 60-lb. wt. loss in 8 mo. Nervous for yr.	Almost entirely intrathoracic	Compressed anteriorly	A-P diameter increased. Hyperresonant. Prolonged expiration. Wheezes.	+49%	Thyrotoxicosis cured. Progressive emphysema with wheezing and cough during next 7 yr.

perspired freely and had preferred cold weather "for years." The rest of the history was non-contributory. Physical examination revealed a tall, obese, nervous male who was 74 inches in height and weighed 228 pounds. The temperature was 98.6, pulse 110, respirations 28 and blood pressure 160 mm. of mercury systolic and 100 diastolic. There was slight bilateral exopthalmos. A fine tremor of the hands was present. The skin was warm and moist. The thyroid gland showed very slight enlargement of the right lobe on palpation; there was no bruit. The chest was emphysematous; the anterior-posterior diameter of the chest was enormous, measuring 30 cm. Wheezes were scattered throughout the lung fields. The heart was enlarged to percussion. The rhythm was grossly irregular; the rate was 124. There were no murmurs. Slight edema at the ankles was noted. Basal metabolic rate was plus 30 per cent. An electrocardiogram showed auricular fibrillation, no axis deviation and low voltage. The circulation time was 28 seconds (decholin). The vital capacity of the lungs was 3,000 cc. in 8 seconds. X-ray examinations revealed a substernal mass which compressed the trachea and displaced it to the left. The heart was 20 per cent larger than normal by height-weight ratio. After ten days of therapy with Lugol's solution the basal metabolic rate fell to plus 8 per cent.

On March 9, 1945, subtotal thyroidectomy was performed and a hyperplastic right lobe weighing 92 gm. was removed from the retrosternal space. After operation the basal metabolic rate was plus 5 per cent, the heart rhythm became normal and the circulation time was 18 seconds. No further specific therapy was given. Nine months later the patient was readmitted for study because of increasing dyspnea. At that time the heart size was 10 per cent above normal. The blood pressure was 140 mm. systolic and 98 diastolic. The venous pressure was 11 cm. of water and the circulation time was 18 seconds. The vital capacity was 2,300 cc. in 12 seconds. All evidence of hyperthyroidism had disappeared. The lungs showed no congestion. Cardiac catheterization on January 9, 1946, confirmed the clinical diagnosis of chronic cor pulmonale due to chronic emphysema.[1] The mean pressures in the pulmonary artery, right ventricle and right atrium were 57/38, 55/29 and 25 mm. of mercury, respectively, and showed marked respiratory variations. The arterial oxygen saturation was 88 per cent. The cardiac output was reduced, the cardiac index being 2.43.

During the next two years the patient was treated with digitoxin, aminophylline, ephedrine and intermittent intramuscular injections of mercuhydrin. Although still dyspneic when last observed in January 1948, the patient was able to carry on his work as a hospital attendant.

Comment: This patient undoubtedly had a mediastinal goiter for many years. Whether it preceded the pulmonary emphysema cannot be determined. The possibility of mediastinal goiter first came to mind because he was suspected to having masked thyrotoxicosis. The latter diagnosis was considered because of the presence of a warm moist skin, sweating, the preference for cold weather and the finding of auricular fibrillation not explicable on other more common grounds.

Case 12 also demonstrated the difficulties in diagnosis and the complications of tracheal compression:

CASE REPORT

CASE 12.—A 42-year-old woman was admitted to the Peter Bent Brigham Hospital on February 14, 1928, because of extreme exhaustion of four months' duration. She had chronic mild rheumatoid arthritis of seven years' duration. In 1922 a slight enlargement of the anterior neck was noted by the patient, and this had remained unchanged up to the time of admission. In 1926, non-seasonal "asthma" began and it also had persisted to admission. A chronic cough and episodes of "choking" sensations had also been present during those two years. Physical examination revealed a poorly nourished female. The temperature was 98.6, the pulse rate 72, the respirations 18 and the blood pressure 100 mm. of mercury systolic and 70 diastolic. The thyroid gland was diffusely enlarged to a moderate degree. There was no bruit. The heart was normal. The anterior-posterior diameter of the chest was increased. Inconstant wheezes were present throughout the lung fields. The skin was normal. There was slight prominence of the right eye and a right lid-lag. The basal metabolic rate varied from plus 13 to plus 23 per cent. X-ray studies revealed a soft mediastinal shadow and displacement of the trachea to the right. The patient refused operation.

Five years later the patient returned, the condition unchanged, and on March 21, 1933, total thyroidectomy was performed. A very large (250 gm.) hyperplastic substernal thyroid gland was found adherent to the trachea and was removed without undue difficulty. The postoperative basal metabolic rate was minus 8 per cent. The "asthma" and other symptoms were completely relieved. When last observed almost ten years later the patient was in good health except for arthritis. Wheezing and a slight cough had recurred only with upper respiratory infections in the eight years following thyroidectomy.

Comment: In this case the diagnosis of goiter was obvious although the degree of toxicity was so slight that it had been overlooked. Operation was advised and later performed mainly because of the mechanical compression on the trachea. This proved curative.

In another case the patient had masked thyrocardiac disease with emphysema. There was a small substernal goiter which was difficult to demonstrate:

CASE REPORT

CASE 10.—A 55-year-old male was admitted to the Peter Bent Brigham Hospital on January 29, 1948, because of progressive angina pectoris of three years' duration. He used 4 to 12 nitroglycerin tablets daily. Direct questioning revealed that the patient had a slight tremor of the hands which had interfered with writing during the preceding two years. For a year preceding hospitalization, mild intolerance to heat and an increased appetite without gain in weight had been noted by the patient.

Physical examination revealed an alert male with a salmon-pink complexion. The skin of the face appeared distinctly suffused, and that of the rest of the body was warm and quite moist. The temperature was 98°, the pulse rate 80, respiration 16, and blood pressure 140 mm. of mercury systolic and 70 diastolic. The eyes were slightly prominent. The thyroid gland was not palpable. The chest was barrel-shaped and hyperresonant. The breath sounds were diminished. There were a few inspiratory wheezes. A slight tremor of the fingers was present. The basal metabolic rate varied from plus 20 to plus 36 per cent. The blood protein-bound iodine was 10 gamma per 100 cc., considerably above normal. An electrocardiogram showed right bundle branch block. The vital capacity of the lungs was 2,900 cc. in 14 seconds. The circulation time was 18 seconds, and the venous pressure was 115 mm. of water. A posterior-anterior x-ray film of the chest showed emphysema. The routine x-ray report made no mention of substernal goiter. On repeat examination when the roentgenologist was

asked to look for a submanubrial goiter, such a mass was identified.

It was decided to observe whether the anginal state could be controlled with medical therapy. The patient was given 10 drops of Lugol's solution thrice daily for two weeks and 100 mg. of propyl thiouracil thrice daily for two days followed by 50 mg. thrice daily thereafter. The improvement was striking. In two months the patient was able to walk a mile or two without stopping and had no anginal pain. However, subtotal thyroidectomy might yet be advisable to relieve the pressure on the trachea and possibly retard the progress of the emphysema.

Comment: Thyrotoxicosis was suspected at once because of the patient's salmon-pink complexion, slight stare of the eyes and warm, moist skin. It was only then that a history consistent with that diagnosis was elicited. The presence of a mediastinal goiter seemed likely because of the absence of a palpable thyroid gland in the neck and the signs of pulmonary emphysema. On the basis of this clinical concept, repeat x-ray studies were made which demonstrated the substernal mass.

COMMENT

The 13 cases reported herein are by no means all that were observed at the Peter Bent Brigham Hospital. Many similar cases of emphysema occurred in which the gland was obviously visible in the neck. In others in which the patients had mediastinal goiter the evidence of emphysema was less striking. These 13 were selected as outspoken examples to illustrate the clinical features under discussion.

In all patients with definite thyrotoxicosis or goiter the possibility of tracheal compression ought to be studied. The authors are impressed with the high incidence of pulmonary emphysema in patients with goiters, although it is difficult to prove a casual relationship in some cases. If hyperthyroidism is suspected the presence of a substernal mass has to be considered, particularly when little or no enlargement of the thyroid can be detected in the neck (Cases 3, 4, 10, 12, and 13). Finally, in all patients with emphysema the possibility of mediastinal goiter causing tracheal compression has to be entertained. Such a goiter may be the cause of many otherwise unexplained instances of dyspnea and decreased vital capacity.*

When the degree of tracheal compression is slight, pulmonary emphysema may develop gradually over the course of many years without subjective complaints (Cases 4 and 10). The clinical symptoms which should direct attention toward this possibility are the suffused face of the patient, prolongation of expiration, an increase in the anterior-posterior diameter of the chest and other objective signs of obstructive emphysema. X-ray examination may be quite helpful but is not conclusive. A large mediastinal mass is readily detected; a smaller one can easily be overlooked on routine examination unless the roentgenologist is asked to look for it. Some may be elusive even when searched for. The actual pressure effect on the trachea may or may not be obvious. It is not unlikely that a slight degree of constant pressure, difficult to visualize by roentgenologic measures, may be productive of a chronic emphysematous state.

Surgical removal is obviously the only treatment for a non-toxic goiter which is producing compression of the trachea. But alternative methods of treatment with antithyroid substances or radioactive iodine are now available in the treatment of toxic goiter. Such non-surgical methods of therapy should not be used unless careful investigation shows no mechanical interference of the trachea. If the goiter is substernal or producing tracheal compression and/or distortion it should be removed surgically in order to prevent or arrest irreversible, progressive bronchopulmonary disease.

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*The following experience is a telling illustration of some of the above considerations. One evening at a conference of several physicians the value of vital capacity determinations was being discussed. Some of the men present thought that its main usefulness was in following the progress of patients with cardiac disease once the diagnosis had been established. The authors insisted that at times it was also helpful in leading to fundamental diagnoses. At this point one of the physicians said that for many years he had had a low vital capacity (3,200 cc.) and yet had no heart disease and felt quite well. In rebuttal, thinking that this particular individual should normally have a vital capacity of about 4,500 cc., the authors stated that if his heart were normal there was reason to suspect conditions like emphysema, mediastinal goiter or other mediastinal masses. The interesting point was that this individual promptly replied that some ten years before a large mediastinal goiter had been removed.

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Results of Medical and Surgical Management in Ten Cases of Congenital Megacolon

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SUMMARY

Of 20 patients under 15 years of age with proven diagnoses of megacolon, ten were treated surgically with either partial colectomy or resection of the entire colon down to the rectosigmoid junction after thorough trial of medical management. Follow-up shows five of these patients as 100 per cent relieved, one as 75 per cent relieved. Three were entirely well a few months after operation but have not been heard from since. One died of peritonitis on the 16th postoperative day.

In four cases in which lumbar sympathectomy was done, the result was partial, temporary or no improvement.

Of the patients not operated upon, several have been lost to follow-up, some are doing well on medical treatment, one died in early infancy, and resection is being considered for two.

For children with congenital megacolon the authors recommend first a thorough trial of medical treatment consisting of diet, vitamins, drugs and enemas. This should be started as soon as possible after the diagnosis is made, in an attempt to prevent the distention and hypertrophy of the bowel from progressing. If distention remains after a reasonable trial period and the child is not gaining weight adequately, requires repeated hospitalization, and is three years of age or more, then resection of the affected portion of the colon is indicated. The risk of operation has been somewhat reduced with better supportive measures and chemotherapy now available. Since infants and extremely young children do not stand operation on the colon as well as older children, decision to operate should take into consideration the age of the patient. In the reported series, the patient who died following operation was the youngest—2½ years of age.

MEGACOLON, or Hirschsprung's disease, is of relatively rare occurrence but is seen in a medical center often enough to require establishment of a definite regimen for its control. In the last 12 years at the University of California Hospital, there have been about 20 proven cases of this condition in children under 15 years of age. Ten of these patients have had either partial colectomy or resection of the entire colon down to the rectosigmoid junction, and these will form the basis of this report. The first six have been reported upon in more detail previously.[11] Several of those not operated upon have been lost to follow-up; some are doing well on medical treatment; one died in early infancy; and resection for two is being considered.

This entity has been recognized since Hirschsprung's description 60 years ago. However, the exact cause of the disease has not been determined as yet. It is fairly well established that there is an imbalance of the autonomic nervous system controlling bowel function. The result of stimulation of the parasympathetic system is to increase peristalsis and relax the internal sphincter, whereas stimulation of the sympathetic nerves will inhibit peristalsis and tighten the sphincter. The beneficial results obtained with the use of the counteracting drugs, Mecholyl (sympathetic stimulant) and Syntropan (parasympathetic depressant), as well as the results from lumbar sympathectomy in megacolon, support the imbalance theory but shed no light on the cause of the imbalance. Present knowledge is limited to the fact that in some cases the disease is caused by underactivity of the parasympathetic nerves, while in others overactivity of the sympathetic nerves is the cause.

At the time of the earlier report,[11] the reports in the literature, with the exception of those of Ladd and Gross,[7] and Dixon,[2] mentioned resection of the colon in megacolon only to condemn it. Since then several observers — Whitehouse,[10] Grimson,[4] and Herrmann[5]—have advocated the operation for patients who do not respond to medical treatment. There are some investigators who still feel that lumbar sympathectomy is the operation of choice. In four of the cases in the authors' series, this operation gave partial, temporary or no improvement. It is conceded even by those who advocate the operation that sympathectomy results in a greater risk of later volvulus and perforation, even though the immediate result may be good. It does not alter the

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hypertrophied colon or the redundant mesentery. There is also the consideration of probable sterility in the male.

The symptomatology and findings in the ten cases of megacolon observed by the authors varied somewhat in degree but they had these points in common:

1. Severe constipation, starting either from birth or within the first few months;
2. Failure of ordinary laxatives or suppositories to produce normal bowel movement, and the necessity of repeated enemas;
3. Increasing abdominal distention;
4. Malnutrition (all but one case);
5. Absence of demonstrable obstructive lesion;
6. Marked dilatation of a portion or all of the colon on roentgenographic study.

Careful diagnostic procedures were carried out before operation was considered. The child was hospitalized for thorough cleansing of the bowel. This often required several days, as it was exhausting for both the patient and the nurse. It required 32 liters of solution at one session for partial cleansing of one 13-year-old child. When the abdomen was flat and the colon relatively clear, x-ray studies were made, with Umbrathor used for the contrast medium, because it is much easier to remove than even a thin barium suspension. Sigmoidoscopic examination was done to rule out obstructive lesion. Some of the patients received spinal anesthesia but since little in the way of useful information was obtained, it is not used routinely any longer.

Medical treatment was begun by placing the child on a high caloric balanced diet with added vitamins, especially vitamin B; mineral oil was given to soften the stool. Mecholyl, which is a parasympathetic stimulant, was given one hour after breakfast in an initial dose of 50 mg., and gradually increased up to tolerance or effect. Some patients received as much as 600 mg. daily. In contrast to the results reported by some observers, several of the patients treated by the authors had moderately severe reactions such as vomiting, sweating, decrease in pulse rate, and (in one case) collapse. These reactions were readily controlled with atropine. Mecholyl[8] has been the most satisfactory of the drugs although Syntropan[6] has been given a trial in most of the patients.

After the diagnostic procedures were done and the medication started, most of the children were sent home for a trial on the medical regimen. When it was decided by the pediatric and surgical staffs that adequate improvement had not been obtained on the medical program, resection was advised. Penick[9] considered medical treatment a partial failure if more than one enema a week was necessary. The authors' criteria for determining the success of medical treatment were the general well-being of the child and the control of the abdominal distention. Many of the children had daily bowel movements but the stools were small, and as the abdomen remained distended the movements were interpreted as overflow and not true evacuations. It was recognized that the giving of medicines and attention to bowel habits and diet was more than some of the mothers could manage. The age of the patient was also a definite factor in determining treatment, since infants and young children do not stand operation on the colon as well as older patients. The youngest patient in the authors' series, 2½ years of age, was the only one who died.

The surgical procedures varied somewhat from the earlier to later cases. In the first case a Mikulicz operation was done, since the patient had a volvulus of the sigmoid at the time of operation. The next group had cecostomy followed by resection of the involved portion of the colon. In most of these patients there was a definite line of demarcation between normal and affected bowel. One patient required another operation three and a half years later for resection of the ascending colon. Because of this, the last two patients in the series, in whom the whole colon was found to be involved, had a one-stage operation with resection of the entire colon down to the rectosigmoid junction. In none of the ten patients was there involvement of the rectum, so that a normal segment was available for the distal anastomosis. Grimson[3] pointed out that medical management of a patient with a normal rectum is difficult and that there is great danger of impaction and perforation. He also advocated operation for this group.

Prior to operation the child was admitted to the hospital for a thorough cleansing of the bowel, a course of sulfasuxidine (when it became available), and correction of anemia if that was present. Sulfonamides were given parenterally after operation, and in the last two cases penicillin was used. The blood proteins were maintained by plasma, if necessary. Fluids, electrolytes, and vitamins were given parenterally. The postoperative course was not always smooth, and some of the children lost a great deal of weight. However, convalescence was rapid once they were on their feet again. One patient (Case 2 in Table 1) gained 35 pounds in one year after a second operation. Her last visit to the clinic was as an adolescent girl of 14 who wished a diet to reduce her hips; no reduction of the abdomen was needed.

The appearance of the colon at operation was remarkably similar in all patients. There was dilatation of the affected colon to many times the normal size. The wall was thickened, soggy, and lifeless. The appearance has been likened to that of thick wet leather. The taeniae and haustra were absent. The sigmoid and its mesentery were usually elongated, a factor predisposing to volvulus. Microscopic examination showed the wall to be hypertrophied and edematous with lymphoid hyperplasia throughout. In most cases myenteric nerve plexi were prominent. As it is hard to conceive of the return of the bowel to normal after such hypertrophy, even considering the excellent regenerative power of children, it would appear to be advisable to check the process at the stage of dilatation and before hypertrophy is reached.

Postoperative roentgenographic studies in several cases have shown evidence of an enlarged colon.

TABLE 1.—*Treatment and Results in Ten Cases of Megacolon*

Case	Involvement Shown by X-ray	Mecholyl	Sympathectomy Age	Year of Operation	Resection	Age	Results
1. M.	Mid-transverse to distal ½ sigmoid	No	Bilateral at 7½ years	1936	Sigmoid colon	12	Excellent at present (24 years)
2. F.	Whole colon; rectum normal	No	No	1939 1942	Mid-transverse to sigmoid Complete colectomy	5 8½	Excellent at present (14 years)
3. M.	Whole colon; rectum normal	Fair results	Bilateral at 3 years	1941	Left colon to rectum	4½	Excellent at present (10½ years)
4. M.	Sigmoid	Poor results	No	1942	Sigmoid	3½	Excellent—6 mo. postoper. No report since
5. F.	Sigmoid	No response	No	1942	Sigmoid	4	75% relief on Mecholyl (10 years)
6. M.	Left colon; rectum normal	No response	No	1942	Left colon to rectum	6½	Excellent—10 mo. postoper. No report since
7. M.	Whole colon; rectum normal	No response	Bilateral at 4¼ years	1944	Sigmoid	4½	Excellent immediately. No report since
8. M.	Left colon; rectum normal	No response	Left - 2 years Right - 4 years	1945	Left colon to rectum	13½	Excellent at present (16¾ years)
9. M.	Whole colon; rectum normal	Some response; not adequate	No	1947	Complete colectomy	5	Excellent at present (5¾ years)
10. M.	Whole colon; rectum normal	Some temporary response	No	1947	Complete colectomy	2 years 7 mos.	Died on 16th postoper. day

but without clinical difficulty. As yet it is too early to be certain that there will not be recurrence of the old difficulty in these cases. The status of the patients 20 to 30 years from now will give the answer. At present the longest follow-up in the series is 12 years.

As may be seen in Table 1, there were 10 children in the series, ranging in age from 2½ to 13½ years at the time of the colon resection. Roentgen examination showed three patients (Cases 1, 6 and 8) had involvement of the left half of the colon; two patients (Cases 4 and 5) of the sigmoid alone; and five patients (Cases 2, 3, 7, 9 and 10) had involvement of the entire colon. All but the first two were given mecholyl for a trial period before operation, some with partial but inadequate response. Bilateral lumbar sympathectomy was carried out in four patients (Cases 1, 3, 7 and 8) with either temporary or no response. Eight had partial colectomy (one required a second operation for more complete resection three and a half years later). Three patients had resection of the entire colon to the rectosigmoid junction; one of them died on the 16th postoperative day of peritonitis from perforation of the rectal stump. Follow-up shows five patients as 100 per cent (mothers' evaluation) relieved, one as 75 per cent relieved; three were entirely well a few months after operation but have not been heard from since, although before the operation their parents had not hesitated to get them to the office when that was considered necessary, despite the difficult transportation during war years.

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Dysplasia of the Hip: A Factor in the Development of Static Osteoarthropathy

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SUMMARY

Dysplasia of the hip is associated with a shallow acetabulum. It may be present and unrecognized until middle age when arthritic changes develop as a result of repeated microtraumata. By that time the process is irreversible.

It can be recognized in infancy by roentgenographic study of the hips, and corrective measures can be taken to develop a normal hip and so prevent the crippling arthritis of later life. Although it is not certain that dysplasia develops in every person having a shallow acetabulum, this structural abnormality is known to be a strongly predisposing factor and therefore should be corrected.

DYSPLASIA of the hip is associated with a shallow acetabulum (Figure 1a). Any discussion of this subject must include the congenital dislocated hip, since the mechanism is the same for the two, except that in the congenital form the dysplasia has not reached the degree of true dislocation. Whether the fault lies in the failure of development of the head or of the acetabulum is still a disputed point. It is not the purpose of this paper to enter into the controversy as to the cause of dysplasia in congenital dislocation except to mention briefly the various theories.

The earlier investigators considered the origin of dysplasia of the hip to be on a mechanical basis, that is, that a relaxation and elongation of the surrounding ligaments and articular capsule permitted the head to dislocate and that lack of the stimulus to osseous growth around the acetabulum provided by the head resulted in hypoplasia of the ilium. The extent of this mechanism (according to the theory) would determine whether the head rested partly in a shallow acetabulum or would be completely dislocated.

In the past 30 years there has been another concept in which the primary fault is considered to be an inherent factor in the development of the acetabulum. Hart,[3] one of the exponents of this belief, has stated that the principal anatomical feature is the acclivity of the roof of the acetabulum which is increased over the normal. The hereditary factor, Hart concluded, is in a dominant hip dysplasia gene carried over from father to son. He based this conclusion on observation of several cases in which one member of a family had a congenital dislocation and others in the same family had a flat socket. He also noted that a patient with congenital dislocation of one hip might also have a flat socket in the other. Faber[2] also came to the same conclusion. He made a roentgenographic consanguinity study on members of the families of ten children with congenital dislocation of the hip and found that there were no dysplasias in the family on the maternal side while in the family of the father four cases of hip dysplasia without dislocation were found. In some of the families only a few individuals had a dislocation while many showed the hip dysplasia without dislocation, the dysplasia being three times more frequent than the actual dislocation.

Badgley[1] in 1943 disagreed with this view. He expressed the belief that the primary cause is interference with normal rotation of the limb buds and that in the dysplasias there is an increased anteversion of the head and neck. He pointed out that normally during fetal life an anteversion develops from 0 in the first two months to 35 degrees at birth. After birth the anteversion should diminish. Normally the increasing pressure on the back of the socket stimulates the deepening of the socket and increased growth on the posterior rim of the acetabulum. This stimulus lacking, the posterior rim does not develop and there is a gradual migration of the head posteriorly. The changes of the acetabulum are secondary and accommodative to the primary failure of rotation. Therefore, Badgley pointed out, these changes are correctible even after birth in the first year of life by maintaining internal rotation and abduction to aid the forces of internal rotation.

Whatever the cause, the fact remains that there is a close relationship between dysplasia of the hip, a flat acetabulum, and a congenitally dislocated hip. Since by abduction and internal rotation in infancy, preluxated hips can be made normal, early recognition of preluxation is important. The congenital dislocated hip will be recognized when the child

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begins to walk, but in the child with a flat socket changes in the hip will develop as the head adapts itself to the shallow acetabulum. Because of the inclination of the roof, the head in adapting itself will become large, broad and flattened, the neck short and wide, and coxa vara will develop. In the final stage it is often confused with healed slipped epiphysis or healed Perthes' disease. This will be

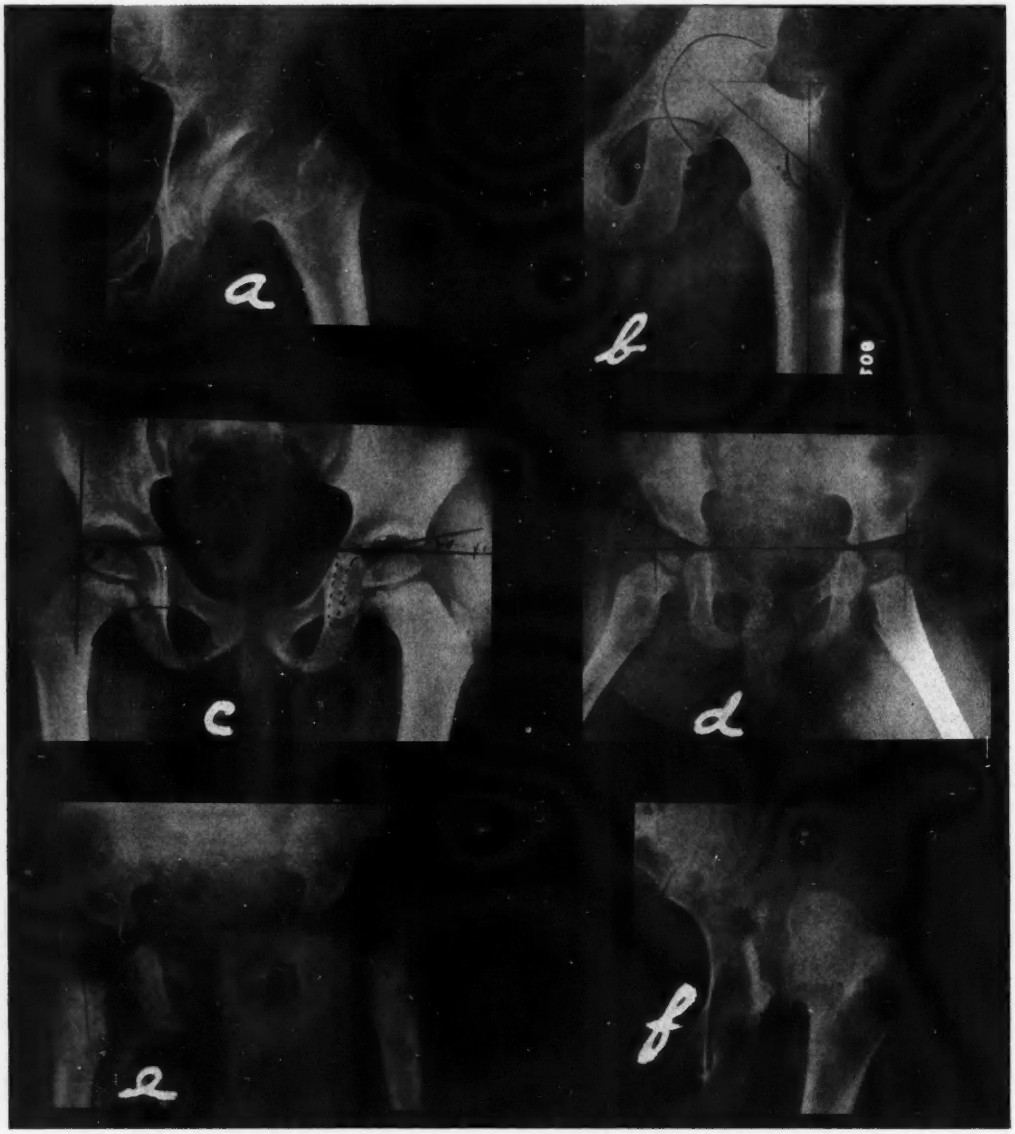

Figure 1.—(a) Shallow acetabulum. The acetabulum is shallow with only a small segment of a curve where a small portion of the head fits into the acetabulum. The head is enlarged, the neck is shortened; the shaft rides high in relation to the neck and is externally rotated.

(b) Normal. Acetabulum is a circular segment of about 170°. Upper border is horizontal. Two-thirds to three-fourths of head fits into acetabulum. Axis of neck forms an angle with shaft of about 130°. Line through fossa of head at right angles to axis of shaft extends through greater trochanter. Continuous curve between inferior margin of neck and inferior margin of pubis. "Shenton's line."

(c) Normal development of acetabulum. (1) Y line through clear space in acetabulum. (2) Vertical line through lateral border of acetabulum. (3) Head of femur lies in medial lower quadrant. (4) Roof of acetabulum forms angle less than 20° with Y line. (5) Shenton's line. (6) Teardrop. Formed by floor of acetabulum and long border of the small pelvis.

(d) Inclination of the roof of the acetabulum is greater on the right side than on the left. It measures 33°; the other side measures 13°. The epiphysis of bone head lying in normal position. The head on the right is not as fully developed as the left.

(e) The acetabulum on the right measures 30°. The shaft is in normal relation to the acetabulum. The acetabulum on the left measures 45°. The head is luxated, lying in the outer lower quadrant.

(f) Congenital dislocation. The acetabulum is flat. The head rides in a false acetabulum in the wing of the ilium.

discussed later. It is inevitable that in middle life traumatic arthritis will develop from this poor mechanical structure because of the continuous pressure on the hip and frequent repeated microtraumata. Wiberg[5] in 1939 supported this view by presenting roentgenograms of 19 patients with primary hip dysplasia before and after development of secondary changes of osteoarthitis.

As groundwork for a study of changes of the dysplastic hip, consideration should be given first to the normal hip (Figure 1b). In the normal hip the head fits into the acetabulum for about two-thirds of a circle. The acetabulum is a circular segment of about 170 degrees, and the upper border tends to be horizontal. The axis of the neck meets the line of the shaft at an angle of about 125-130 degrees. Another line extending through the fovea of the head at right angles to the shaft will cross the greater trochanter.

The development of the acetabulum can best be studied in the child before union of the parts takes place (Figure 1c). In the normal hip joint of the newborn the acetabular cavity is divided into two equal parts by the Y line. This Y line is a prolongation of the horizontal line in the clear space between the two segments of the acetabulum which has not united. If a vertical line is dropped from the outer rim of the iliac roof of the acetabulum to the Y line, this region is divided into four right-angled quadrants. Normally the epiphysis for the head of the femur lies in the medial lower quadrant. As the child grows, the Y-shaped cartilage alters its direction so that the socket is divided into a smaller upper segment. The acclivity of the roof of the acetabulum as determined by the angle it makes with the Y line ranges from about 12 degrees to 30 in the newborn. This angle gradually decreases as the child grows until it becomes almost horizontal in the adult. The depth of the acetabular cavity is shown by the configuration known as the teardrop. This is not an anatomical structure but a shadow on a roentgenogram made up of three lines—a small curved line, a very long almost straight line, and a line connecting the two. These three lines form a drop resembling a tear and were first pointed out by Kohler,[4] who gave the figure its name. The arch of the tear figure is the contour of the acetabular cavity and corresponds to the acetabular wall. The small semicircular arch corresponds to the part of the bone where the anterior opening of the acetabular fossa curves around backward to the wall of the pelvis. The third straight line is the shadow of the wall of the pelvis running in the direction of the ray.

Two factors are involved in dysplasia of the acetabulum. One is the curve. Instead of the normal segment of a circle of about 170-175 degrees, the arc will be less than 170 degrees. The upper part of the roof, instead of being directed horizontally, is directed upward so that the angle with the Y line is increased. The greater the upward direction, the more pronounced the flatness of the acetabulum—up to 180 degrees as is sometimes found in the congenital dislocated hip (Figures 1d, e, f). It is the flattening that gives the tendency for the head to slip upward out of the fossa.

The other factor is the depth of the acetabulum. This can be determined by the shape of the previously mentioned teardrop shadow on a roentgenogram. The shallow acetabulum is manifested in increased width of this teardrop.

With the development of dysplasia there is also a retarded development of the epiphyseal nucleus of the femoral head on the side that is involved. Treatment early by abduction and internal rotation will often restore the acetabulum to normal (Figures 2a, b, c, d).

Patients with a dysplastic hip may go through early life absolutely free of symptoms. Others in early adult life may complain of a feeling of fatigue and sometimes they may limp slightly, especially after unusual exercise. This was especially noted in a military training camp where many men who had never before complained of hip pain reported to the dispensary after being subjected to long hikes. The complaint was hip pain and fatigue. In most cases, clinical examination disclosed no abnormality. Diagnosis can be made with certainty only with roentgenograms. In many cases there was a considerable degree of dysplasia without symptoms and the condition was accidentally discovered in the course of examinations made because of other complaints. However, as previously mentioned, although the condition may cause little or no distress in young persons, it is almost inevitable that in middle life the secondary changes of traumatic arthritis will develop because of the repeated microtraumata as shown by a loss of joint space, sclerosis, cystic areas of rarefaction, and many times a double acetabular wall with osteophytic deposits (Figures 2e, f).

As was noted in an earlier paragraph, the late changes in dysplasia of the hip may be confused with healed slipped epiphysis or healed Perthes' disease.

Perthes' disease usually occurs between the ages of three and twelve (figure 3a). Symptoms, including a limping, are always present and some patients have pain. It is a focal aseptic subchondral necrosis of the neck and the neighboring epiphyseal center of the head. It starts first as a rarefaction of that part of the metaphysis bordering the epiphyseal line, with the bone being replaced by fibrous tissue and cartilage. The process progresses into the interior of the head of the shaft, developing subchondral bone necrosis. The necrotic bone becomes fractured by weight-bearing, with a resulting deformity of the surface contour. Repair is slow, fibrous tissue replacing the bony fragments. The head becomes compressed and flattened, which accounts for the term coxa plana. There is also compression and expansion of the proximal end of the diaphysis. Traumatic arthritis at the site of the lesion is a late development (Figure 3b).

Slipped epiphysis, also known as adolescent coxa vara, begins in adolescence. It is found in children,

in boys predominantly, who are overweight for their age and height and who have flabby, weak skeletal musculature and more than a normal amount of subcutaneous fat. Because of the abnormalities, some observers believe that endocrine disturbance may be an etiological factor, or at least that there may be a constitutional predisposition to a condition of this type. At first, in the pre-slipping stage, there is a

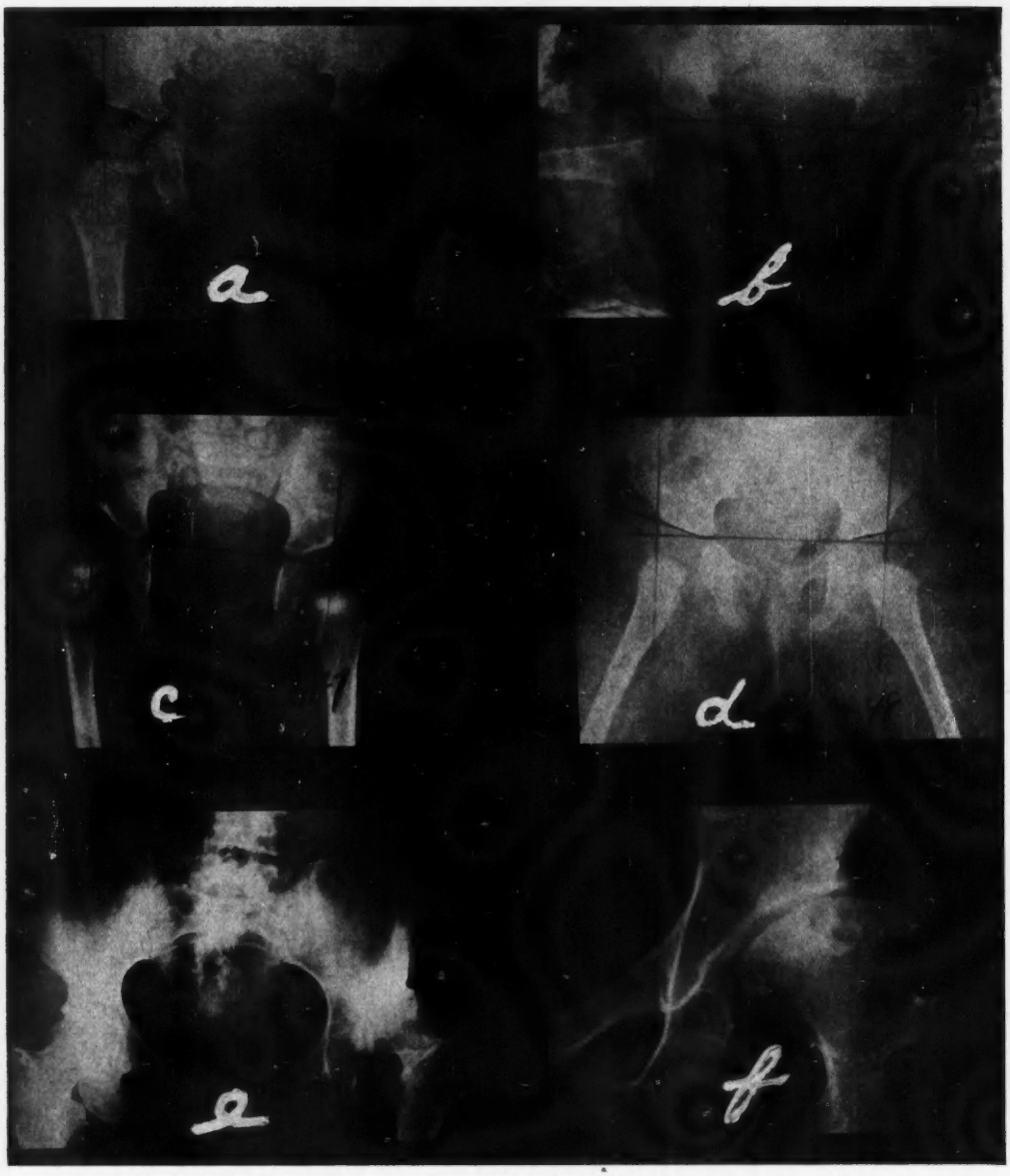

Figure 2.—(a) The acetabulum on the right measures 22°. The head lies in normal relation. The left acetabulum measures 41°. The head lies in the upper outer quadrant. Stage of congenital dislocation.

(b) Two months after treatment. The acetabulum on the right measures 21°. The acetabulum on the left measures 34°. The heads of both femora lie in the acetabula. Both femora have been abducted and internally rotated.

(c) The acetabulum on the right measures 34°. The epiphysis for the right femur lies in the outer inferior quadrant. The left measures 28°. The epiphysis for the head lies in the inferior inner quadrant.

(d) Six months after treatment. The heads of both femora are in normal position, lying in the inferior inner quadrant. The epiphysis for the right is delayed in relation to the left. The acetabulum on the right has reduced from 34° to 18° and the left from 28° to 15°.

(e) Shallow acetabulum on the left side with sclerosis of the acetabulum and the head of the femur showing traumatic arthritic changes.

(f) Shallow acetabulum with cystic changes in the head of the femur indicating early arthritic changes.

widening of the epiphyseal line, decalcification, and irregularity in density of the juxta-epiphyseal strip of the femoral neck (Figure 3c). Often there is history of trauma, usually of a trivial nature. It is noted, however, that the head retains a normal position in the acetabulum and becomes displaced backward on the neck, the epiphysis being rotated posteriorly (Figure 3d). There is a gradual ascent of the femoral neck in the acetabulum (Figure 3e). Unless reduced, the malformation will lead to degenerative

Figure 3.—(a) Early Perthes' disease. Broadening of the diaphyseal line. Destructive areas in the head as result of an aseptic necrosis.

(b) Old healed Perthes' disease with subsequent traumatic arthritis.

(c) Slipped epiphysis—early stage. Widening of the diaphyseal line of the right hip with slight rarefaction of the diaphyseal end of the neck.

(d) Slipped epiphysis—early stage. Lateral view shows an inward and slight backward rotation of the head of the femur.

(e) Old slipped epiphysis with an inward rotation of the head, widening of the diaphyseal line, and a broadening of the neck, resulting in a coxa vara deformity.

(f) Old slipped epiphysis with resultant traumatic arthritis.

arthritis of the hip joint (Figure 3f). Such a joint usually becomes painful and functionally useless by the time the individual reaches middle age, whereas in dysplasia symptoms may not start until middle age. It is noted that both in Perthes' disease and in slipped epiphysis the depth of the acetabulum may be normal, and that there is a history of the disease starting in childhood.

COMMENT

Because it can be corrected in early life, dysplasia of the hip is a challenge to the pediatrician to discover the lesion early and so institute preventive treatment; to the radiologist who must acquaint himself with this syndrome; to the orthopedist to treat properly the infants who have the disease and so establish a normal hip socket; to the general practitioner who must recognize in the young adult a dysplastic hip and so advise the patient to take an occupation which will subject the hip to minimal trauma in the hope of preventing arthritis later.

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Clinical Application of Electrokymography

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SUMMARY

The electrokymograph may be used in getting information to augment clinical manifestations of abnormalities of cardiac motion. It is adaptable to the study of other activities in the body such as respiration and the movement of masses.

In electrokymographic examination of four patients with constrictive pericarditis with calcifications, the authors noted a distinctive wave pattern common to all. Similar waves were obtained from localized areas in three patients with suspected constrictive pericarditis.

Differences between the waves produced by pulsatile aneurysms and those of non-pulsatile masses adjacent to the cardiac silhouette were noted in experimental studies.

UNTIL recently, the best known methods of obtaining records of the motion of the heart with the thorax intact have been roentgencinematography[3] and roentgenkymography.[10, 14] In 1945, Henny and Boone[8, 9] introduced a third roentgen method, electrokymography.

The electrokymograph is an instrument designed to detect and record variations in the intensity of a small beam of x-rays. It consists of a photoelectric tube with fluorescent material covering the photosensitive side (this combination being called the "pick-up unit"), an electrocardiographic apparatus as a recording device, and a fluoroscope. The photoelectric tube is activated by x-rays falling on the fluorescent material, and the resulting electrical current is led to the electrocardiographic apparatus. Variations in the intensity of the x-rays are translated into variations in electrical current, and recorded as a line on photographic paper which is marked with lines indicating time intervals. An increased flow of current, due to increase in the intensity of the x-rays reaching the fluorescent material, causes the line being traced on the paper to be directed downward; a decrease in current causes the line to be directed upward.

The pick-up unit is mounted between the patient and the fluoroscopic screen. Using the fluoroscopic screen, the unit is positioned so that the center of its fluorescent surface is opposite the border of the heart. When the heart border moves laterally, as it does in diastole, more of the heart lies between the sensitive surface and the x-ray tube, reducing the intensity of the x-rays reaching this surface. This causes the line being traced on the photographic paper to move upward. Conversely, in systole, with less of the heart between the sensitive surface and the x-ray tube, the line moves downward.

When the pick-up unit is directly over the heart, rather than opposite a border of the heart, all of the x-rays of the primary beam which reach the sensitive surface pass through the heart. The fluctuations in x-ray intensity recorded are due to variations in thickness of the heart during the cardiac cycle. The heart is thickest in diastole, hence the intensity of the x-rays reaching the pick-up unit during diastole is less than during systole. Tracings obtained in this manner are called "density tracings." They closely resemble the tracings obtained from the heart border; during systole the line moves downward, and during diastole it moves upward.

To permit identification of the phases of the cardiac cycle on the electrokymogram, the carotid pulse is simultaneously recorded on the same photographic paper, using a pressure capsule fastened over the carotid artery on the patient's neck.

NORMAL CURVES

Adequate descriptions of normal electrokymograms of the heart and great vessels may be found in the literature.[1, 2] Hence, the authors have included only a resume of the general appearance of the normal curves. Examples are shown in Figure 2.*

As will be recalled, cardiac systole extends from the time of closure of the auriculoventricular valves to the closure of the semilunar valves (aortic and pulmonic). Systole has two principal phases: (1) isometric contraction, during which the ventricle is a closed chamber in which the pressure rapidly rises; (2) systolic ejection, beginning when the semilunar valves are forced open because the intraventricular pressure exceeds the pressure in the aorta and the pulmonary artery, and ending with the closure of the semilunar valves. Diastole also has two principal phases: (1) isometric relaxation, in which the ventricle is again a closed chamber; and (2) the

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*This presentation is arranged so that references to the literature will be incorporated with each condition described. The sections on constrictive pericarditis, intrathoracic masses, and in part the section on paradoxical motion represent original reports. The remaining sections cover material studied by the authors but also reported previously in the literature. It will become obvious to the reader that complete detail concerning each of the conditions can not be reported in this paper.

filling period, beginning with the opening of the auriculoventricular valves, and continuing to the closure of the auriculoventricular valves.

The carotid pulse tracing provides two landmarks for identifying the phases of the cardiac cycle in the electrokymograph. The foot of the major ascending limb of the carotid pulse marks the beginning of the systolic ejection phase. The tip of the dicrotic notch on the descending limb indicates the closure of the semilunar valves, since it is produced by the impact against the valves of blood moving toward the ventricle.

The onset of systolic ejection is recorded instantaneously on the photographic paper, since the mechanism for this is entirely electrical. In recording the carotid pulse, however, it takes approximately 0.02 second for pulse waves to travel from the level of the aortic valve to the carotid artery, and 0.01 second for its transmission from the pressure capsule over the carotid artery to the recording device. One must measure back, therefore, from the foot of the major ascending limb of the carotid pulse, a distance corresponding to 0.03 second, to find the point on the heart tracing at which systolic ejection begins.

Through the use of the carotid pulse, the onset of systolic ejection can be identified on the ventricular electrokymograph. Systolic ejection is usually recorded as a short upward movement of the tracing followed by a steep downward movement, indicating a brief lateral movement followed by a greater medial movement of the ventricle. The end of systolic ejection and the onset of the isometric relaxation phase of diastole are identified by the dicrotic notch on the carotid tracing, the same time correction used for systolic ejection being necessary.

Within approximately 0.1 second after the beginning of isometric relaxation, as determined from the dicrotic notch, the electrokymograph is seen to ascend rapidly at first, and then more slowly as it reaches the full height of the curve. The initial rapid ascent is due to lateral movement of the ventricle wall as the ventricle fills rapidly after the opening of the auriculoventricular valves. The slow ascent corresponds to slower filling after the initial rapid emptying of the auricle. At or near the top of the electrokymographic curve, diastole usually ends abruptly. A short descending line follows, representing the period of isometric contraction. It ends at the point which marks the onset of systolic ejection, completing the cycle.

The short waves produced as the result of isometric relaxation and contraction frequently are referred to as secondary waves superimposed on the large fundamental waves associated with ventricular filling and emptying. These are due to rotation and change in shape of the heart, especially noticeable during the isometric periods.[1, 2]

The commonest auricular electrokymogram consists of two waves: the first peak occurring at the onset of the major ascending limb of the carotid and the second peak at or just following the carotid dicrotic notch. The ascending limb of the first peak corresponds to ventricular filling and isometric contraction; the descending limb represents motion of the auricle towards the midline due to ventricular contraction. The ascending limb of the second peak begins at or near the crest of the carotid pulse wave and represents the filling of the auricle which pushes its borders laterally, overcoming the medial pull of the ventricle; the descending limb begins at or very shortly after the closure of the semilunar valves and is related to isometric relaxation and possibly to early ventricular filling. Less commonly but certainly not infrequently, three waves are seen in the auricular electrokymogram. The two basic waves described above are present; the third one consists of a short downward movement on the ascending limb of the first wave. This is associated with auricular systole.

Graphs of the aorta and pulmonary artery are similar in configuration to pressure and volume tracings obtained by direct methods. Both the aorta and pulmonary artery produce waves paralleling the carotid pulse wave, with time differences corresponding to their distance from the ventricles.

In order to illustrate the potentialities of this instrument in fields other than the cardiovascular system, electrokymograms of diaphragmatic motion and pulsation in the lung fields have been included in Figure 1.

Electrokymograms from the cardiac silhouettes of individuals with apparently normal cardiovascular systems vary considerably in their detail; however, there is a remarkable constancy in their fundamental configuration and time relationships within relatively narrow limits. Even though the "normal" pattern may vary from individual to individual, and variation can be detected between records obtained at different points on the same chamber of the same individual, the normal electrokymograms contrast quite sharply with records obtained from clinically abnormal hearts. While it is not within the scope of this presentation to cover the entire field of cardiac abnormalities, a few of the more common abnormalities are briefly presented:

Ventricular Asynchronism. In 1946 and 1947 Ellinger, Gillick and Boone[4] studied the time relationships of motion of the right and left ventricles with those of the ascending aorta and pulmonary artery, and compared these relationships with the carotid pulse wave. In this study it was shown that, to establish the time relationship of the ejections of the right and left ventricles, it is sufficient to measure the time relationship between the onsets of the major ascending limbs of the carotid pulse wave and of the pulmonary artery electrokymograph. These observers found that ejection from the two ventricles does not always occur simultaneously in the so-called normal individual and that the limits of asynchronous ejection in the normal was plus or minus 0.03 second. Asynchronous ejection in excess of 0.03 second is almost always accompanied by electrocardiographic evidence of bundle-branch block.

Figure 2(f) shows an example of asynchronism of ejection with the left ventricle leading. It will be

noted in this figure that the pulmonary artery follows the carotid pulse tracing. The electrocardiogram in this individual showed a right bundle-branch block. By comparison, illustration (c) of this same figure shows the normal relationship of pulmonary artery electrokymograph to carotid pulse tracing; here the carotid follows the pulmonary artery by approximately 0.02 second.

Katz,[12] and Wolferth and Margolies[19] have reported studies which substantiate the electrokymographic findings noted here.

Abnormal Rhythm. Although several methods exist for the determination of the various arrhythmias, it is felt that additional information may be obtained concerning the mechanics of cardiac motion and thus indirectly the quality of myocardial contraction through the study of the arrhythmias. Figure 2(a, b, c, e, and f) shows examples of several of the more common types of abnormalities associated with rhythm.

Paradoxical Motion. Paradoxical motion of an ischemic area of myocardium has been the subject of several publications. Tennant and Wiggers[17] in 1935 produced coronary occlusion in localized areas of a dog's heart and by means of direct myographs demonstrated paradoxical motion in the area. Hirsch,[11] Faber and Kjoergaard,[5] Gubner and Crawford,[7] Sussman, Dack and Master,[16] Thomas and Garland,[18] and others, have repeatedly demonstrated by means of the roentgenkymograph a reversal of motion in certain areas of the ventricular wall in

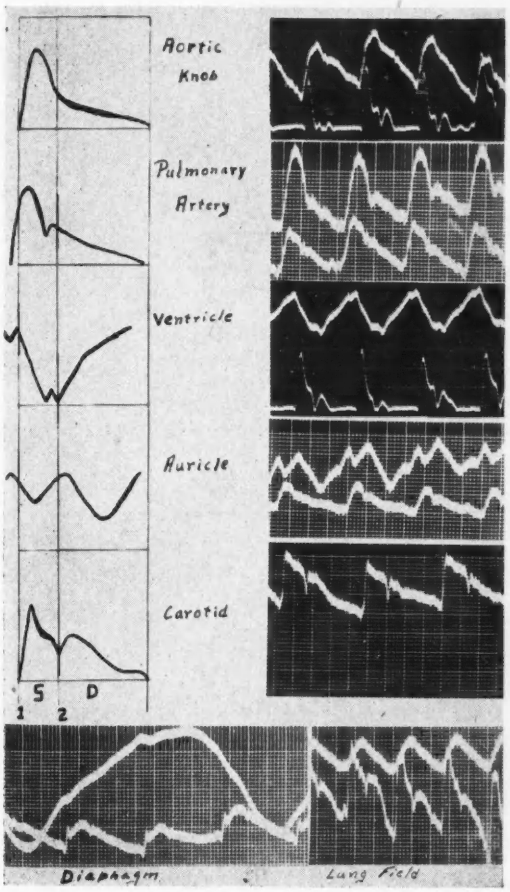

Figure 1.—Schematic and actual records from selected areas of the cardiac silhouette of normal individuals. Ventricular record of the mid-left ventricular border on a postero-anterior projection; auricular record of the mid-right border of the cardiac silhouette on a postero-anterior projection. Right dome of diaphragm. Lung field was from the fourth intercostal space (anteriorly) at the mid-clavicular line. S=systole; D=diastole; 1 marks onset of major ascending limb of carotid; 2 marks tip of dicrotic notch. (Pulse tracing of carotid artery is below electrokymograph.)

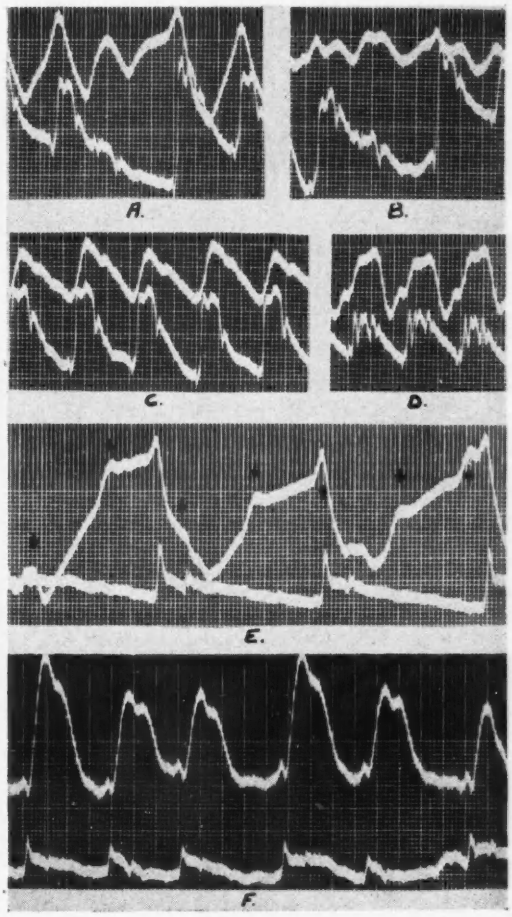

Figure 2.—Selected abnormalities: (a) ectopic beat, at the left ventricular border; (b) ectopic beat, at the right auricular border; (c) alternans as shown on the pulmonary artery; (d) paradoxical motion of the left ventricle in a patient with hypertension and symptoms of coronary insufficiency; (e) complete A-V dissociation as obtained at the mid-left ventricular border (auricular waves indicated by arrows); (f) electrokymogram of pulmonary artery illustrating both auricular fibrillation and delayed right ventricular ejection—carotid precedes the pulmonary artery by .04+ second. (An electrocardiograph tracing showed a right bundle-branch block.) (Pulse tracing of carotid artery is below electrokymograph.)

individuals in whom clinical evidence of extensive myocardial infarction had been noted. Recently, Murray[13] reported similar observations on the hearts of dogs. Electrokymographic examination of individuals who have given definite clinical evidence of extensive myocardial infarction have revealed abnormal motion in areas of the ventricular border, and in some instances the curves obtained closely resemble those reported by Tennant and Wiggers.[17] Figure 2(d) shows an example of paradoxical ventricular motion.

It is of interest to note that occasionally similar curves have been obtained in individuals with cardiac complaints but no definite electrocardiographic evidence to support a diagnosis of infarction or severe myocardial ischemia.

Constrictive Pericarditis. Electrokymographic examination of four patients with radiologically and surgically proven constrictive pericarditis with calcification has produced a constant type of ventricular curve. The ventricular wave has the appearance of a "square wave"—that is, its top is flat and it is practically devoid of secondary waves. The descending or emptying component of the wave has about the same duration and angle as the ascending or filling limb. The over-all appearance of several cycles is that of a straight line with V-like interruptions constituting the emptying and filling components. Figure 3 illustrates ventricular electrokymograms obtained from three of these individuals.

It is of interest to note that similar waves have been obtained from localized areas in three subjects with suspected constrictive pericarditis.

A similar type of wave can be reproduced by placing a water-filled balloon inside a container which limits the filling of the balloon but does not interfere with its emptying. Figure 3 illustrates an electrokymogram of such a balloon.

Intrathoracic Masses. The differential diagnosis of abnormal shadows in the area of the cardiac silhouette has been most perplexing. The roentgenkymographic approach has been marked by encouragements and disappointments.[6, 15] The authors approached this problem by setting up a crude model simulating a vessel with an aneurysm and a heart with a spherical tumor adjacent to it. Both "vessel" and "heart" were supplied with sufficient expansile force so that motion would be applied to both the "aneurysm" and the "tumor." Density and border electrokymographic studies were made.

As may be seen in Figure 4, the experimental "aneurysm" was found to expand synchronously with the "vessel" in all border and density positions.

Figure 5 diagrammatically shows the experimental "tumor" or "mass" and the points at which it was studied for border and density changes. Motion was imparted to the mass by motion of the "heart." Unlike the expansile "aneurysm," the slit (photosensitive surface) of the electrokymograph was covered to a greater extent along the distant border

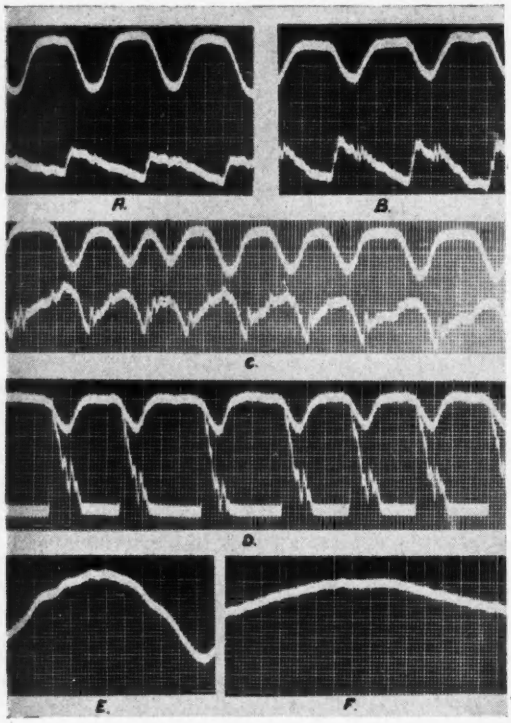

Figure 3.—Square waves obtained from the cardiac silhouette of three patients with clinical and radiologic evidence of constrictive pericarditis proved by operation. (a) and (b) are left and right ventricular electrokymograms respectively, from the same patient; (c) mid-left ventricular border—also shows auricular fibrillation; (d) mid-left ventricular border in a patient operated upon for constrictive pericarditis in 1937 with recurring evidence of constriction and operative intervention in 1948; (e) and (f), electrokymograms of a balloon; (e) represents free motion; (f) represents restricted motion of the same balloon due to cardboard collar. (Pulse tracing of carotid artery is below electrokymograph.)

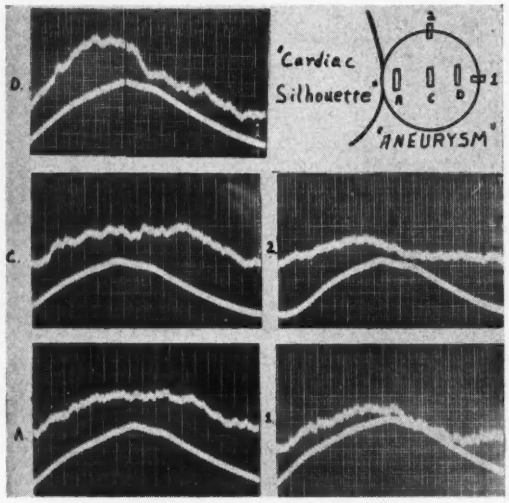

Figure 4.—Experimental "aneurysm" showing direction of motion at areas indicated on diagram. The upper curve is the electrokymograph of the "aneurysm"; the lower curve represents the direction of motion of the moving force (heart or great vessel).

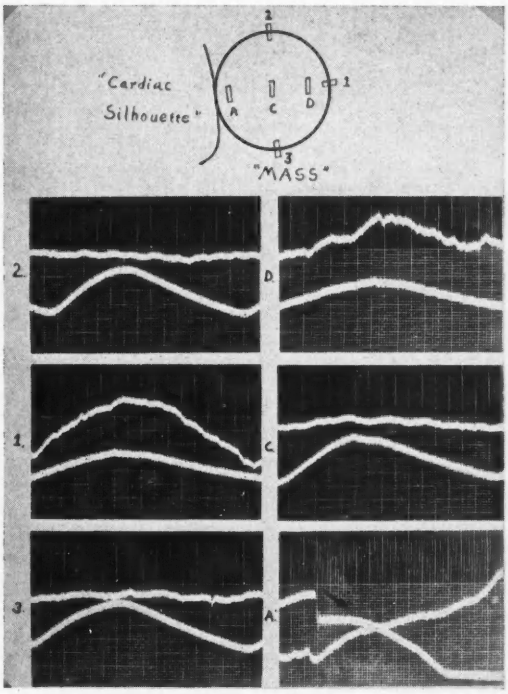

Figure 5.—Experimental "mass" adjacent to the cardiac silhouette. "Cardiac silhouette" represents the portion of the heart or great vessels transmitting motion to the mass. Positions D and 1 show the electrokymographic curve and the curve from the moving force—both move in the same direction; position A shows the two motions going in opposite directions; other positions show no or only indefinite electrokymographic motion. (Arrows are drawn to and in direction of the tracing from the moving force which represents either the heart or great vessels.)

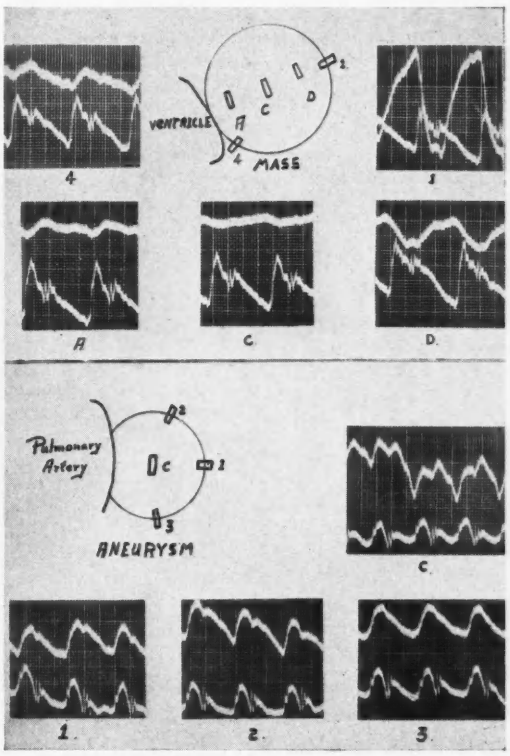

Figure 6.—Actual electrokymograms taken from a dermoid cyst (proved by operation) adjacent to the left ventricle and from an obvious aneurysm of the pulmonary artery. Numbers indicate areas from which corresponding records were obtained. (Pulse tracing of carotid artery is below electrokymograph.)

while the adjacent border was uncovered, thus producing waves in opposite directions. Electrokymograms from borders parallel to the line of motion showed little or no change. Density changes at the center of the "mass" during motion were of such small magnitude as to produce little or no electrokymographic change; as the slit was moved from the center toward the periphery along the line of motion, the changes were more marked. Density changes adjacent to the "heart" produced waves opposite in direction to the heart motion while in the distant periphery the changes were in the same direction as the "heart." The rationale for these observations will be made in a separate report.

Similar observations were made on selected patients with known tumors or aneurysms, and the results were found to be consistent. An example of each is shown in Figure 6.

It must be emphasized that this is a brief summary of the electrokymograph and its uses and that the discussions of constrictive pericarditis and the differentiation of aneurysms and intrathoracic masses are preliminary reports. The study of these conditions is still in progress. Nevertheless, an appraisal of the electrokymograph at its present stage of development is desirable.

It represents a valuable extension of the fluoroscopic technique, providing a permanent record of the movements seen in fluoroscopy, available for study after the examination has been completed, for restudy, and for comparison with subsequent examinations of the same patients. In effect, it is a fluoroscopic "microscope," demonstrating movements too small in amplitude, or too short in duration, for the unaided eye to perceive. It permits correlation in time of movements occurring in separate parts of an organ, too far apart for the eye to correlate accurately, or of movement seen with the patient in two or more different positions.

A comparison of the electrokymograph with the roentgenkymograph as to the advantage and disadvantages of each would seem appropriate. At present it appears that the former has at least the following advantages: the movements as recorded are amplified so that smaller movements are brought out and larger ones are more easily studied; a larger number of cycles can be recorded; areas to be studied can be selected fluoroscopically, in their optimum projections; the slit can be aligned in the direction of the motion of the part; correlations in time of movements of various parts of an organ are more accurate.

There are, on the other hand, these disadvantages: Measurement of true amplitude of movements is not as yet possible with the electrokymograph; the precise point along the border of the part at which the movement is recorded is not shown as it is on the roentgenkymogram (this problem, however, can be remedied by the use of a spot film device in conjunction with the electrokymograph); more cooperation on the part of the patient is required; the procedure is more time-consuming; it is necessary that there be in the fluoroscopic room a technician trained in the use of the apparatus, although this training can be very quickly acquired. A most important disadvantage is the greater possibility of overexposure of the patient to radiation.

It may be that the electrokymograph will be used much as the "spot film" is used in gastro-intestinal work—it can be immediately brought into play to secure for the record a phenomenon which the fluoroscopist has seen on the screen, or to enable him to study at his leisure a movement he does not understand, with the assurance that this particular movement, at this particular point, will be recorded for him. The roentgenkymograph, like the large gastro-intestinal film made in the radiographic room, will provide him with an over-all view of the motion of the part, and may indicate which areas require further and more detailed studies.

Should a choice between the roentgenkymograph and the electrokymograph be necessary, the authors are of the opinion that the electrokymograph, due to its greater flexibility in use and clarity of graphic records, would be the more valuable instrument from both the clinical and research points of view.

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Discussion by S. F. THOMAS, M.D., *Palo Alto*

It is rarely these days that modern medicine produces something which is almost entirely new. The electrokymograph is a modification of the use of the fluoroscope and in the terms of Drs. Gillick and Reynolds, it is a "fluoroscopic 'microscope.'" As usual, with most electronic gadgets and most mechanical improvements in medicine, the method is more complicated than anything that preceded it, but it has many advantages which balance most of the difficulties.

It is not easy to pick up even the rationale of some of their discussion. However, with considerable time and just a little use of the instrument, one quickly becomes acquainted with the excursions and while roentgenkymography offers a slightly confusing background, it does give one some notion as to the movements that are being recorded.

Roentgenkymography and electrokymography could probably be found to supplement one another, but undoubtedly the electrokymograph is a finer instrument than the roentgenkymograph, and if eventually some way of calibrating the electrokymograph is found, the mechanical advantages of the roentgenkymograph will disappear.

The uses of the electrokymograph are found in the arrhythmias, and some of the finer points of cardiophysiology have already been explained by the electrokymograph which could be explained in no other manner.

In this day of ever-increasing death rate from degenerative diseases, insults to the myocardium are more and more frequent; it is my belief that some of the insults (silent occlusions and/or ischemias) to the myocardium cannot be recorded with the usual electrocardiograph, and a mechanical means of some sort of recording cardiac movements will be found helpful in prognosticating the significance of some chest pain and debatable electrocardiographic findings. The roentgenkymograph acts to corroborate the electrocardiograph and close some of the loopholes in electrocardiography. The electrokymograph, being of still finer sieve, will undoubtedly close still further some of the gaps in the screen of the usual electrocardiogram or roentgenkymograms. Drs. Gillick and Reynolds are most conservative and have claimed but little for the electrokymogram along these lines.

The most striking advancement with the electrokymograph is the differential of aneurysm vs. solid tumor in the chest where it is often desirable to differentiate between pulsating and non-pulsating masses. This should prove very valuable and the experimental results obtained so far, which are more than preliminary in nature, are most encouraging.

The Papanicolaou-Traut Method of Cancer Diagnosis

Its Use as a Routine Pathologic Laboratory Procedure

STUART LINDSAY, M.D., *San Francisco*

SUMMARY

In a series of 450 examinations by the smear technique in the pathologic laboratory of a small private hospital, the total diagnostic error on all smears was 6.6 per cent. The error on vaginal smears was 4.7 per cent. These figures include false positives, false negatives, and all those smears classified as suspicious even though positive or negative diagnoses were made on subsequent examinations.

The Papanicolaou-Traut method of cancer diagnosis can easily be made one of the routine pathologic procedures in the small hospital laboratory. Technicians with little previous cytologic experience can be trained to screen vaginal and other smears accurately after a short training period. This cytologic method is proving to be of value in the early detection of some neoplasms, and if its application is to be extended, the practicing pathologist should add the test to his diagnostic routine.

IT IS generally agreed that further reduction in the mortality from malignant disease depends at present largely upon early diagnosis. The careful study of Papanicolaou and Traut[5] on the cytologic diagnosis of uterine cancer has initiated the use of a diagnostic method by which early malignant processes may be detected. In addition, it has the advantage that repeated examinations may be made without the disadvantages of minor surgical procedures. Knowledge of this technique has been extended by the reports of Meigs and his co-workers,[4] Jones,[3] Ayre[1] and others. Gates and Warren[2] recently presented an extensive appraisal of the method. At present there seems to be increasing enthusiasm for and acceptance of this method of diagnosis as a screening procedure and as an adjunct to other diagnostic tests in selected cases of malignant disease.

In general, pathologists have not shared in this enthusiasm, and to date few pathologists have used and evaluated the method. Since there is no fundamental difference between observation of malignant cells in sections of tissue and in smear preparations, and since pathologists often use cellular criteria alone in the microscopic diagnosis of malignancy in tissue sections, this diagnostic adjunct would seem to belong within the field of pathology. Certainly, pathologists should be able to learn this cytologic method of diagnosis more quickly and easily than those less familiar with cytologic and histologic details. Furthermore, if the use of the method is to be extended to screen significant numbers of the population in the cancer age group, the possibility that pathologists and their laboratory assistants could assume part of this task should be considered.

With few exceptions,[6] the recorded experience with this method has been in the laboratories of teaching centers and large cancer clinics, where research and the training of physicians and technicians have been a large part of the cytologic program. The reporting of experience and evaluation from smaller hospital laboratories seems warranted, since, as the method becomes more widely used, these large teaching centers obviously cannot continue to cope with the problem as a routine diagnostic test.

It is the purpose of this presentation to report on the use of the Papanicolaou-Traut method of cytologic diagnosis as a routine procedure in the pathologic laboratory of a small private hospital. This diagnostic method as applied to vaginal and other fluids appears to be entirely feasible in the small laboratory in a small community. In a period of seven months a number of unsuspected neoplasms were discovered. A total of 450 examinations were made (approximately 900 smears) on 419 patients.

The laboratory concerned in this report is in a private hospital which has 139 beds and which had 43,938 patient-days in 1947. The laboratory employed 11 technicians and performed a total of 32,108 laboratory procedures in 1947. There were 2,119 surgical specimens examined and 85 autopsies performed in 1947. Approximately 50 per cent of the clinical-pathological procedures were done on out-patients. The hospital serves as the medical center of a suburban county of 207,000 people and is situated in a town with a population of 34,000. There are 135 physicians in the county, and the majority are on the hospital staff. Some of the vaginal smears were submitted for study by six gynecologists, but many were contributed by general practitioners, internists and others. The staining and screening were done in the pathologic section of the laboratory by a licensed technician.

During the period from October 15, 1947 to May 15, 1948, this method was made available to the physicians of the community without a charge being made to the patient. The availability of the method was made known to the physicians in the hospital staff bulletin. No charge was made initially for two reasons: (1) It was believed that if physicians were able to obtain smears on many of their patients

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either as a screening or diagnostic procedure, they would be able to evaluate the test in relation to their own practices, and (2) it was felt that further experience with a volume of material by both the technician and the pathologist was desirable before a fee was charged for the examination. It has been agreed that this cytologic test is a screening procedure or a diagnostic adjunct, that it does not replace the biopsy as a means of diagnosis (except in rare instances) and that it should be interpreted in the light of the clinical findings as are the results of other laboratory procedures.

Once the test has been established as a routine procedure, a charge to private patients should be made. The overhead costs can be calculated easily. The fee should be low enough to encourage use of this diagnostic method.

Many of the vaginal smears were taken by physicians as part of a routine physical or pelvic examination on well patients. Some were taken from patients with symptoms presenting diagnostic or prognostic problems. A few were requested by the pathologist on cases of proven pelvic, urinary or suspected pulmonary carcinoma in order to obtain positive smears for study. Several smears from patients with radiated cervical carcinoma were examined. During the seven-month period, all gastric fluids containing no free hydrochloric acid on chemical analysis were examined for malignant cells. The majority of specimens were from out-patients; a few were sent to the laboratory from the county hospital for indigents in the same community. Surgical pathological findings from these patients having cytologic tests were readily available for correlation.

Labeled bottles with tight screw-caps, partially filled with 95 per cent alcohol-ether fixative, are available in the laboratory where they may be obtained by physicians or their office nurses. A supply of mimeographed requisition forms and instruction sheets are given to the physicians with the bottles of fixative. The requisition forms include spaces for pertinent data which may be helpful in interpreting the smears, and the instruction sheets give in detail the steps in obtaining and fixing the smear preparations. These forms are patterned closely after those described by Papanicolaou and Traut.[5] When sputum, urine, ascitic fluid or gastric fluid is to be examined, it is taken to the laboratory and the smears made in an appropriate manner by the technician. Better preparations have been obtained from fresh material than from these miscellaneous fluids received in equivalent amounts of 70 per cent or 90 per cent alcohol. At least two smears are made from the sediment of these fluids. It is requested that two smears be made on vaginal fluid examinations. A significant number of vaginal smears received are unsatisfactory because of scantiness or distortion due to drying. In these instances, the report includes a request that the test be repeated. Some physicians have obtained excellent smear preparations by dispensing with the aspirating tube. They collect the vaginal fluid in the posterior half of the vaginal speculum, and make thin smears with the gloved finger.

The staining technique used is that of Papanicolaou and Traut[5] in preference to the use of hematoxylin and eosin or other counterstains. With the former stain, the excellent nuclear and cytoplasmic detail and the translucent quality make the contrast between normal and abnormal cells more striking than with other staining procedures. Excellent Kodachrome photomicrographs can be made from these smears. Satisfactory stains can be done by inexperienced technicians simply by following the prescribed steps of the staining method. With an average of only four smears received per day, it seems reasonable to stain them only once or twice a week, unless a report is urgently requested. Large staining jars are used for all solutions, and it has been found necessary to discard and replace the staining solutions only at intervals of one to two months.

Widespread use of this diagnostic method depends on the availability of technicians trained to examine the entire slide and mark atypical, suspicious or malignant cells. With only an occasional smear to examine, a pathologist could do the entire examination. However, with a larger number of slides to examine, the average pathologist in a busy laboratory does not have the time required for detailed microscopic searching. In this laboratory the technician was on a 40-hour week and spent approximately half the day preparing tissue sections and the remainder of the day staining, screening and typing reports on the vaginal and other smear preparations. She was able to learn the fundamentals of screening during an informal training period of two weeks in Traut's cytologic laboratory at the University of California Medical School. Even in this short period she became adept in screening the slides and marking any atypical, suspicious or malignant cells for later review and decision by the directing pathologist. With continuing experience, her proficiency is of course increasing. At present, the average single smear can be screened accurately and abnormal cells marked in approximately ten minutes.

In this laboratory, all slides including those reported as "negative" by the technician, are examined by the pathologist, since an occasional significant finding may not be noted by the first observer. It is the pathologist's responsibility to identify the abnormal cells present, and to classify them as "suspicious" or "positive" for malignancy. Obviously this responsibility should not rest with the technician. It is to be regretted that already demands from physicians and patients for the test have led to its performance by technicians and technologists, without supervision, in commercial laboratories. This is a dangerous situation, since these unqualified individuals then are attempting to diagnose malignant disease.

METHOD OF REPORTING RESULTS

Instead of Papanicolaou's method of reporting the cytologic findings as Class I, II, III, IV, V, in this laboratory the terms "negative," "suspicious"

or "positive" are used. That a single negative vaginal smear is not conclusive evidence of the absence of uterine malignancy has been repeatedly stressed to physicians submitting smears. If suspicious symptoms or lesions are evident, the test obviously should be repeated.

On vaginal smears in women past the menopause, atypical (though not frankly suspicious or malignant) basal cells are occasionally encountered. At times, individual cells of this group are almost indistinguishable from malignant elements. In these instances the presence of atypical cells is reported and, following the suggestion of Meigs and his coworkers,[4] a request is made that the patient be given stilbestrol for seven to ten days followed by another smear. Within this period, the cellular pattern becomes largely squamous in type, and the atypical cells disappear. Malignant cells, if present, are not altered by this therapy and are then more easily distinguished from the surrounding squamous elements.

Another source of possible error lies in distinguishing between the cells of hyperplastic endometrium and those of a well-differentiated fundal adenocarcinoma, since there is no sharp line of distinction between them.

Smears are reported as "suspicious" if (a) any cells present show some but not all of the criteria of malignancy, or (b) only a single or small number of definite malignant cells are present. Repeated smears are requested and taken at intervals, and examined until a positive or negative decision can be reached.

The term "positive" is applied only to those smears containing numerous typical malignant cells. It seems evident that conservative reporting will lead to fewer false positive reports.

In addition to the examination for malignant cells, other abnormalities demonstrated in the vaginal smears are reported to the physician. These include the presence of Trichomonas vaginalis, and the degree of estrogenic activity as shown by the relative proportions of basal and superficial cells, particularly if the cytologic picture does not correspond to the patient's age or menstrual history.

RESULTS

Vaginal Smears. A total of 378 vaginal smear examinations were made on 355 patients. With few exceptions, each examination was made on two smear preparations. Of the 378 examinations, 354 were negative for malignant cells. Only one of these was from a patient with proven adenocarcinoma of the uterine fundus. The vaginal fluid was diluted with abundant blood and leukocytes, probably accounting for the failure to demonstrate malignant cells.

There were 16 examinations (32+ smears) in which the cells found were classified as suspicious. This series represents 13 patients.

Of these, one had a second negative smear, and no further examinations were requested by the physician.

Endometrial biopsy on the second patient showed an endometrial hyperplasia with groups of atypical non-malignant cells.

A third patient had two suspicious smears, followed by one smear classified as positive. Subsequent cervical and endometrial biopsy showed only a mild endometrial hyperplasia. Following this procedure, one smear examination gave negative results.

A fourth patient, aged 46, who had menorrhagia, had one examination showing a few suspicious cells plus hyperplastic endometrial cells. Two subsequent examinations showed negative smears.

A fifth patient, aged 22, with a serosanguinous vaginal discharge for several months, showed a few suspicious cells on one examination with two subsequent negative smears.

The sixth patient was 72 years of age and had had several episodes of vaginal bleeding. The first examination showed an atrophic smear with numerous basal cells plus numerous suspicious cellular elements. Two subsequent smears showed atypical cells interpreted as hyperplastic endometrial cells.

The seventh patient, aged 72, had had six months of vaginal bleeding. The initial examination was reported as suspicious and a second set of smears was reported as positive. Biopsy was refused by the patient but clinical signs of definite fundal carcinoma appeared subsequently.

The eighth patient, aged 68, with intermittent vaginal bleeding, had an atrophic smear with one suspicious cell present. Two subsequent sets of smears after administration of stilbestrol (1 mg. per day for 10 days) were negative.

The ninth patient in this group was 25 years of age and was in the sixth month of pregnancy. Two sets of smears were reported as suspicious. Further clinical examination showed a sessile, firm, 1 cm. elevated lesion on the cervix. Microscopic examination of the excised lesion showed cellular pleomorphism and increased mitotic activity of the squamous epithelium of the surface. This did not appear definitely malignant, but may have represented a precancerous lesion. Subsequent smears following local excision of the lesion were negative.

The tenth patient, aged 37, had had vaginal discharge and slight bleeding for one month. Clinical examination of the cervix showed nothing abnormal. Three sets of vaginal smears at intervals of ten days showed one to be suspicious, one negative, and a third to be again suspicious. Reexamination of the cervix showed no definite lesion except for a small bleeding point at one lateral margin of the cervix. Biopsy of this area (followed by fulguration) showed an early, infiltrating, epidermoid carcinoma. Serial sections of the cervix following total hysterectomy revealed no residual neoplastic tissue.

The eleventh patient, aged 56, had had intermittent vaginal bleeding following removal of small endometrial polyps one year previously. On the first examination, the smears contained a few suspicious cells which on five subsequent examinations were repeatedly demonstrated and considered to be hyperplastic endometrial cells.

The twelfth patient, aged 44, had one suspicious smear with no follow-up to date.

The thirteenth patient, aged 48, had no symptoms. Atypical cells were found in the first examination and numerous highly suspicious cells were present in the second examination. Cervical biopsy revealed an early infiltrating epidermoid carcinoma.

Eight sets of vaginal smears on seven patients were classified as positive. The first was from a woman, aged 35, with a recurrent rectal adenocarcinoma and a rectovaginal fistula following an abdominoperineal resection.

The second positive smear was from a 50-year-old woman with irregular vaginal bleeding. Biopsy and total hysterectomy revealed a malignant papilloma of the endometrium (adenocarcinoma-in-situ).

The third patient, aged 33 (the third patient in the suspicious group), had one positive smear. Biopsy of the endometrium and cervix showed no malignant tissue.

The fourth patient, a woman 61 years of age with post-menopausal bleeding, had one positive smear, and biopsy and total hysterectomy revealed a papillary adenocarcinoma of the uterine fundus.

The fifth patient, aged 72 (the seventh patient in the suspicious group), had one positive smear. Biopsy was refused, but definite clinical evidence of fundal carcinoma is present.

The sixth patient, aged 46, had no gynecological complaints or pelvic abnormalities. Two sets of smears were reported as positive, and one of four cervical biopsies taken in the four cervical quadrants and total hysterectomy revealed an early epidermoid carcinoma-in-situ.

The seventh patient, aged 70, had had a sanguinous vaginal discharge for one month. Following one positive vaginal smear (composed almost entirely of typical malignant cells), clinical examination and biopsy disclosed an extensive but superficial neoplasm covering the cervix and vaginal walls.

In summary, then, of 378 vaginal smear examinations on 355 patients, a positive diagnosis was made by smear in seven patients. In six of these, the presence of a malignant process was demonstrated either clinically or by biopsy. There was one false positive diagnosis. Of the 13 patients with 16 examinations, classified as having suspicious cells present, three were subsequently shown to have uterine carcinoma. In one patient, biopsy has not been done to date. Of the other nine patients with suspicious smears, one had a cervical surface lesion with atypical non-malignant epithelium; the remaining eight were not shown to have malignant disease. Hyperplastic endometrial cells and atypical basal cells were among the elements causing suspicion in this group.

Of 354 negative examinations, one was a false negative.

Sputum Smears. A total of 21 specimens of sputum from 14 patients were examined for malignant cells. On one patient with clinical and roentgenological evidence of a carcinoma of the upper lobe of the right lung and with negative bronchoscopic findings, four specimens showed only rare typical malignant cells and these smears were classified as suspicious. A fifth specimen contained definite clumps of malignant cells and was classified as positive. The diagnosis of bronchogenic carcinoma was confirmed later by biopsy of a metastasis and by autopsy. The rest of the smears on the other 13 patients were classified as negative, and these patients were shown subsequently to have non-malignant pulmonary disease.

Gastric Smears. Specimens of gastric fluid from 26 patients with achlorhydria were examined. All smears were negative. One patient had a gastric resection which revealed an infiltrating scirrhous carcinoma.

Urinary Smears. Fifteen single urine specimens from 14 patients were examined by the smear technique. Two were classified as positive; both patients had papillary carcinoma of the bladder. Of four patients with suspicious smears, one had clinical and roentgenological evidence of carcinoma of the kidney with pulmonary metastases, and one had a benign papilloma of the bladder. Of the nine negative smears, three were false negatives. One patient had an epidermoid carcinoma of the bladder and one had an adenocarcinoma of the prostate, both proven by biopsy. An additional patient with a negative smear had clinical evidence of carcinoma of the kidney with skeletal metastases.

Miscellaneous Smears. Miscellaneous fluids (cysts, abdominal, pleural, rectal and prostatic) were examined in only ten patients. There was one positive pleural fluid, later proven by biopsy. All other smears were negative, and there were no false negatives.

Addendum: In the 11-month period since this report was submitted for publication, 700 additional examinations were done on 611 patients. There were 569 vaginal smears examined (505 patients); the remainder were smears of sputum, gastric fluid, urine and other miscellaneous fluids. Of the vaginal smears, 550 were reported as negative, 12 as suspicious and 7 as positive. Of the 131 miscellaneous smears (106 patients), 98 were reported as negative, 15 as suspicious and 18 as positive. The total error on all smears was 5.4 per cent. The total error on vaginal smears was 3.6 per cent.

Grateful acknowledgment is given to Miss Barbara French for the careful technical assistance in this study.

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Contagious Ecthyma in Man

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SUMMARY

The virus of contagious ecthyma of sheep and goats is transmissible to man.

The human infection is a relatively benign, self-limited disease. It is frequently encountered in sheep-raising areas among persons handling infected animals or the vaccine.

Usually only protective treatment is required, but x-ray therapy will produce prompt regression of the skin lesions.

SIX cases, in humans, of a disease which is transmitted from sheep to man and which in humans causes single or multiple skin lesions often accompanied by low grade fever and regional adenopathy, were observed by the author between 1935 and 1947. All the cases originated in Northern California.

The skin lesions were limited to the hands and fingers at a break in the skin as a site for inoculation of the infected material. In each case the patient first observed a small erythematous macule which extended and elevated in the succeeding week. From the histories it was apparent that the macules appeared between one and two weeks after inoculation.

In the second week there appeared a virulent lesion, a sharply circumscribed umbilicated bulla measuring one to several centimeters in diameter and covering a cyanotic reddish-purple edematous bed. Beyond the periphery was a variable-sized zone of erythema. A striking feature in contrast to this vivid lesion was the minimal amount of accompanying local discomfort.

In the cases observed it was noted that at the height of development of the bulla, fever of 99° to 100° might be encountered, together with moderate enlargement and soreness of the regional lymph nodes.

From the second to third week rupture of the bulla, denuding, regression of the underlying bed, and crusting occurred. There was spontaneous recovery in three to four weeks with minimal scar formation barely perceptible after a few months.

Three patients had single lesions (Figures 1 and 2). When multiple, the lesions are seen at various stages of development (Figure 3), and the history given indicated an interval of some days between the initial appearance of each. The oldest lesion was the most advanced, but wide variation in the extent of development or virulence of the several lesions was noted.

From the Department of Radiology, Woodland Clinic Hospital.

Figure 1.—Single benign lesion in second week.

Figure 2.—Single virulent lesion in second week. Mild epitrochlear and axillary soreness.

No reinfection has occurred in any of the six patients. Several had had chickenpox, herpes simplex, or successful vaccination with cowpox; none had had herpes zoster.

Apparently medical advice as to this disease is not often sought unless the lesions develop to an unusual extent. Personal inquiries among sheep workers, and published reports in the literature,[3, 4, 5] indicate that contagious ecthyma is frequently transmitted to susceptible persons handling infected sheep, or the vaccine, and the lesions so acquired may often be minimal in size and virulence. When the infection is minimal and unrecognized, medical advice may not be obtained.

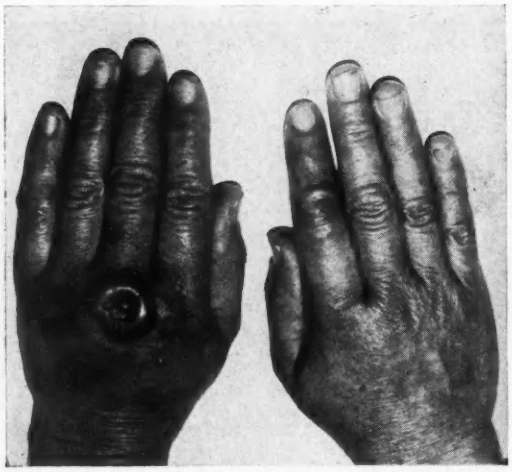

Figure 3.—Multiple lesions. Small lesion, 15 days, left little finger; large lesion, 12 days, left third metacarpal phalangeal area with large zone of erythema extending over dorsum of hand; medium lesion, 7 days, right index finger.

ETIOLOGY

Contagious ecthyma (sore mouth, doby mouth, infectious pustular dermatitis) has been shown to be a disease entity of sheep and goats, caused by a virus which is difficult to filter.[2] It has been known in many sheep-raising countries of the world, but it was apparently first reported in the United States when Howarth described the disease among sheep in California in 1929.[2]

The virus is considered to be quite resistant and may lie dormant in grazing areas for long periods.[1] It produces an eruption of pustules around the mouth, and in non-wool-bearing areas particularly about the head. Immunity against reinfection results and active immunization may be produced by vaccination.[1]

Material from the second patient observed by the author (in 1935) was successfully inoculated into scarified areas on a non-immune sheep, producing pustules as seen in contagious ecthyma.* A similar successful inoculation to sheep from a human lesion was described by Newsome in 1934.[4]

DIAGNOSIS AND TREATMENT

The inability to demonstrate specific organisms in routine bacteriological studies, the history of contact with the virus of contagious ecthyma of sheep, and the self-limited clinical course of the lesion progressing through the macular, papulovesicular, umbilicated bleb or pustular, and crusted stages are considered as criteria for diagnosis.

Treatment consisted of local cleansing and loose dry sterile protective dressings. Three patients at the height of bleb development were given a single medium voltage x-ray treatment (100 r in air). These treated lesions regressed promptly and healed more rapidly than the non-treated lesions.

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*With Dr. J. H. Howarth, Department of Veterinary Science, University of California at Davis, whose untimely death interrupted his immunological studies.

CASE REPORTS

◀ Temporal Arteritis ◀ Charcot's Joint, with Unusual Features, in a Diabetic Patient
◀ Ruptures of the Umbilical Cord with a Case of Intrapartum Rupture of All Three Vessels

Temporal Arteritis

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TEMPORAL or cranial arteritis is a relatively rare disease and the cause is unknown. Horton, Magath and Brown[2] first described this peculiar condition in 1932 and since that time 35 cases have been reported in the literature.

Outstanding characteristics of this febrile disease are painful inflammation of the temporal arteries and the average age incidence of 65, the incidence being greater in women.[4] Pain, usually temporal in location, but sometimes in the occipital and parietal areas when other vessels are involved, low grade fever, general malaise, anorexia, sweating, weakness and loss of weight are prominent symptoms which are present two to six weeks prior to obvious involvement of temporal arteries. Ocular disturbances such as diplopia, photophobia, ptosis and partial or complete loss of vision occur in a considerable number of cases.[1]

Symptoms and course of illness in all cases have been similar and the disease has affected persons in the same age group. The course of this clinical entity is toward recovery within two to six months, although temporary relapses have been noted.[5] No deaths have been reported due directly to the disease and the prognosis for complete recovery is good.

CASE REPORT

The patient, a white housewife, 65 years of age, when observed in the clinic November 25, 1946, complained of very severe headaches which had begun three weeks previously and shortly after recovery from acute coryza. Up until the present illness the patient had enjoyed exceptionally good health all her life, although there was a history of moderately severe dental caries since 1939.

There was excruciating head pain over both temporal areas and both sides of the face, but it was more pronounced in the right temporal region, and there was also moderately severe pain in the occipital area. The patient had been taking opiates every two to four hours for relief of pain.

Ten days before reporting to the clinic, she had begun to experience difficulty in opening her mouth because of pain in the temporomandibular joints, and she was unable to wear a hat due to sensitiveness over the forehead and temporal areas. She had lost ten pounds in three weeks, and there was almost complete loss of appetite. The patient complained of severe lassitude, fatigue, general malaise, night sweats, dullness of intellect, fever and nausea of three weeks' duration.

Examination: Physical examination revealed marked tortuosity, swelling, redness and tenderness along the course of both temporal arteries, more pronounced on the right side (see Figure 1). There was tenderness over both temporomandibular joints, the occipital area and cervical spine.

The vessels in the fundi of the eyes were tortuous, but there was no edema of the discs or evidence of hemorrhage. No visual impairment was noted. Many teeth were absent and several carious teeth present with evidence of pyorrhea of the gums. The tonsils were slightly enlarged.

From The Scripps Metabolic Clinic, La Jolla, San Diego.

Figure 1

Laboratory Findings: At the time of admission to the clinic, erythrocytes numbered 4,140,000 with hemoglobin value of 75 per cent; leukocytes numbered 9,500 with 79 per cent polymorphonuclears, 16 per cent lymphocytes, 2 per cent monocytes, 1 per cent eosinophils and 2 per cent basophils. The blood sedimentation rate was 60 mm. in one hour. Platelets numbered 400,000 per cubic mm. Analysis of catheterized urine revealed a faint trace of albumin, 4 to 6 leukocytes per high-power field and a rare erythrocyte. No growth was observed upon urine culture. Agglutination for Malta fever, typhoid and paratyphoid was negative. Fasting blood urea was 35 mg., fasting blood sugar 92 mg., uric acid 3 mg., and total serum proteins 5.87 per 100 cc. Results of a Kahn test were negative and no growth was observed on a blood culture.

X-ray examination of the skull was reported as follows: "There is a thinning of the temporal bone along the course of the left anterior diploic vein. This is probably due to varicose dilatation of this vein. The left sphenoparietal sinus, into which the above vein empties, is also more prominent than usual. The bones of the skull otherwise have a normal appearance. The sella turcica is normal in size and shape." The right temporomandibular joint had a normal appearance by roentgenological examination. X-ray examination of the cervical spine revealed "narrowing of the sixth cervical interspace and moderate hypertrophic

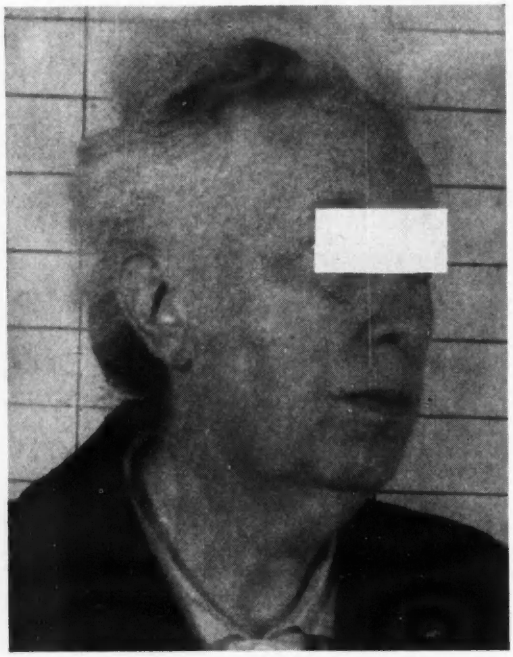

Figure 2

arthritic changes throughout the cervical spine." The chest was roentgenographically normal.

Basal metabolic rate was +12 per cent, and an electrocardiogram revealed left axis deviation.

Course in Clinic: Pain was constant and severe, and frequent administration of opiates was required for relief. At 8 p.m. on the second night following admission, the patient's temperature was 101.8° F. On the assumption that a systemic infection might be involved in this condition, penicillin in dosages of 40,000 units every three hours was begun at this time, and the following morning at 8 o'clock the temperature was recorded at 99.4°. Thereafter, it continued below 100° and down to normal for the remainder of the period of hospitalization. Sodium salicylate 1.2 gm. with sodium bicarbonate 0.2 gm. thrice daily was started on the third day after admission and continued for three weeks. X-ray therapy to each temporal area was given twice the first week of hospitalization.

A total of 2,240,000 units of penicillin was administered. On the fourth day the pronounced signs of inflammation along the temporal arteries were subsiding, although the tenderness and severe headaches continued. After two and one-half weeks there was less severity and frequency of head pain and also diminution in tenderness over the temporal regions. From this time until dismissal from the clinic on January 15, 1947 (see Figure 2), there was gradual improvement with slow subsidence of temperature to normal.

In spite of supplemental iron and vitamin therapy, frequent recheck examination showed a decrease in erythrocytes to 3,640,000 and in hemoglobin to a value of 64 per cent within four weeks. This was attributed to the fact that the patient could not take a well balanced diet due to nausea.

The patient was seen at frequent intervals after leaving the clinic, and the remaining diseased teeth were extracted April 29, 1947. The last erythrocyte count, June 26, 1947, was 4,700,000, with a hemoglobin value of 90 per cent; leukocytes numbered 7,450, and the differential count was normal. There were no head pains or fever and both temporal arteries were normal upon examination. The patient had resumed her usual activities.

COMMENT

In this case the process apparently subsided before the temporal arteries became thrombosed. Biopsy of the affected vessels was deferred due to the early signs of improvement in the patient's condition. As a rule, the course of this illness seems to be unaffected by treatment of any kind. Study of microscopic sections from biopsy segments of involved arteries reveals the condition to be one of chronic periarteritis and arteritis.[3]

SUMMARY

A case of temporal arteritis has been reported because of the rarity of the disease and the scarcity of reports of such cases in the literature.

Outstanding characteristics of this febrile disease are painful inflammation of the temporal arteries and the average age incidence of 65, the incidence being greater in women.

Prominent symptoms such as pain, low grade fever, malaise, anorexia, weakness and loss of weight are present usually two to six weeks prior to obvious involvement of the temporal arteries.

The course of illness is toward recovery, with a duration of two to six months, and it seems to be unaffected by treatment of any kind.

No deaths have been reported due directly to the disease and the prognosis for complete recovery is good.

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Charcot's Joint, with Unusual Features, in a Diabetic Patient

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AS IT was not until recent years that Charcot's joint, or so-called neuropathic, "trophic," degenerative bone lesion, was shown to occur in connection with diseases other than syphilis, the literature contains few reports of cases except those of syphilitic origin.

The case to be reported is one of Charcot's joint in a diabetic patient who, although he had had syphilis, apparently had been cured of that disease several years before, without recurrence.

The largest single group of cases of Charcot's joint in diabetic patients was reported from the New England Deaconess Hospital by Bailey and Root[1] in 1947. They reported 17 cases of lesions of bones of the foot in patients with diabetes, some of whom had demonstrable disease of the

nervous system. In 1948, Hodgson, Pugh and Young[3] reported from the Mayo Clinic 61 cases of degenerative lesions of bones of the foot. In 19 of these cases the patients had diabetes alone, and in them no disease of the nervous system was demonstrated. Other reports of one or two cases are on record. Foster and Bassett[2] in 1947 presented a comprehensive discussion of the possible pathogenesis, along with reports of two cases.

CASE REPORT

The patient, a white male, 38 years of age, was admitted to Mercy Hospital, San Diego, for 14 days in April 1948. After several months in the Army in 1943 he had been discharged, apparently for a variety of reasons, including inability to stay awake. Diabetes had been diagnosed later and had been under fairly adequate control since then. About seven months before admission to hospital, the patient noted rather sudden pain in the first two toes of the left foot, along with swelling and tenderness. He continued in his occupation of bartender, however, and about a week later a small piece of bone extruded through a small draining sinus that had appeared at the base of the second toe. After about three months the foot improved and was fairly usable until March 1948, when swelling and tenderness recurred. Diabetes meanwhile had been fairly well controlled with daily doses of about 30 to 35 units each of protamine zinc and regular insulin. The last known blood sugar level before hospitalization was 176 mg. per 100 cc. four months previously. There had been an occasional minor reaction to insulin. Urine tests done by the patient had shown an occasional trace of sugar. Diabetic cataract had been diagnosed in 1945.

In 1935 syphilis had been diagnosed and intensive treatment had been given in the U. S. Army and Navy Hospital in Hot Springs, Arkansas. The patient had been discharged as cured but subject to follow-up, which had been done periodically since, with blood and spinal fluid being found normal.

Upon physical examination in the hospital it was noted that the left lower extremity was swollen almost to the knee, with swelling diminishing upward from a maximum in the region of the ankle, where pressure left a pit in the flesh. With the patient standing, there was rather marked cyanosis of the foot distal to a point proximal to the metatarsophalangeal joints. Although cyanosis disappeared rapidly with the patient recumbent, the swelling remained. Pulsations at all points were normal to palpation. Thermocouple studies of both lower extremities showed no significant variations.

Extensive varices of the left thigh and leg, involving both the long and short saphenous systems, were noted with the patient standing. The site of extrusion of the bone spicule was noted at the base of the left second toe but the sinus was closed. The foot was somewhat splayed and thickened through the metatarsal arch. Besides the previously mentioned cyanosis, the skin at the swollen area had the "dried apple" appearance of skin stretched over edema. No gross disturbance of sensation of touch, pain or position could be demonstrated.

There was a cataract on the lens of the right eye. The fundi were approximately normal with no retinopathic changes noted. The pupils reacted normally to light and to accommodation tests, and external ocular motions were normal. Marked caries in the teeth and generally poor oral hygiene were noted.

Blood pressure was 145 mm. of mercury systolic and 70 mm. diastolic, but the cardiovascular system otherwise was apparently normal. Hemoglobin was 13.5 gm. per 100 cc., and leukocytes numbered 12,450 per cmm. Blood sugar rose to as high as 216 mg. per 100 cc., blood urea was 29 mg. per 100 cc., and the uric acid content of the blood was 4.6 mg. per 100 cc. Serum calcium and serum phosphorus values were, respectively, 9.0 mg. and 3.9 mg. per 100 cc., and alkaline phosphatase was 2.3 Bodansky units. The ratio of serum albumin to serum globulin estimations was 2.0 gm. to 5.4 gm. per 100 cc. on one occasion, while on another it was 3.8 gm. to 3.9 gm. Results of a cephalin flocculation test were within normal limits, as were also the results of bromsulfalein and serum Kline and Kahn tests.

Spinal fluid cell count, colloidal gold and Wassermann reaction were normal. The protein content of the spinal fluid was 95 mg. per 100 cc.

Because of the abnormal levels of protein in the serum and spinal fluid, and to further the search for a possible cause of the bone lesion, a smear of bone marrow was examined. The normoblast-myeloid ratio was 1:5, and in 500 cells examined nothing abnormal was noted.

Roentgenograms of both hands, both feet, and the chest revealed only low-grade osteoarthritic changes in the right foot, involving the metatarsophalangeal joint of the great toe, dissimilar radiologically from the changes in the left foot, which were as follows: There were degenerative, destructive and proliferative changes involving both the phalanges and the metatarsals. The metatarsophalangeal articulation of the great toe was completely disorganized, with a dispersal of calcerous material in the soft tissues. Similar changes, although less pronounced, were noted in the metatarsophalangeal articulations of the second toe. There was a fracture of the distal end of the proximal phalanx of the fourth toe and a fairly well marked area of destruction in the medial aspect of the head of the fifth metatarsal bone. Diffuse osteitis or subperiosteal calcification was noted along the margins of the first, second and third metatarsal bones. There were similar changes in the proximal phalanx of the second toe.

A patch test showed no tuberculin sensitivity, and when 0.1 cc. of coccidioidin test material was administered intracutaneously, no reaction was noted after 72 hours.

COMMENT

Since it has been shown that Charcot's joint can occur in connection with other diseases than syphilis, diabetes being one of them, it appeared that this case fell into that category, especially as there was no discoverable evidence of syphilis (of which the patient apparently had been cured several years previously) or of syringomyelia, another cause of such lesions of the so-called neuropathic variety. The exact nature of this disease of the bone and its pathogenesis are not clear, but it is obvious that profound changes of the body economy accompany it. In the case reported, there were moderately severe diabetes, lenticular degeneration of the right eye, reversal of the albumin-globulin ratio of the serum (the authors were unable to find reference to this phenomenon in any report of a case of the disease) and pronounced elevation of the spinal fluid protein content with no other changes of the spinal fluid. With relation to the spinal fluid protein content, the experience of other observers has been as follows: Jordan[4] reported from the Joslin group that in 40 cases in which tests were done, the total protein content in the spinal fluid was above 50 mg. per 100 cc. in 22 cases, and the highest level in any case was 120 mg. Bailey and Root[1] reported an average of 69 mg. per 100 cc. in their series, with 100 mg. recorded in two cases and 112 mg. in one.

In the case reported herein, the absence of accompanying neurologic changes attributable to the diabetes is to be noted. The significance of the varices in the leg above the affected foot is debatable, but in this regard the absence of varices in the other leg is at least worthy of note. Whether or not there is significance in stasis of circulation in com-

bination with other factors present is a subject for conjecture.

The objections of Hodgson, Pugh and Young[3] to considering these bone lesions in diabetic patients and in other patients in their series as antedating the soft tissue injury which always accompanies them, apparently are well based. However, their denial of a disturbance of nerve supply, either somatic or autonomic, seems not well founded, since there appears to be abundant evidence, clinical and experimental, that damage to the nervous system can and does produce such lesions—whether primarily in the bone or in the soft tissue being beside the point. The Guillain-Barre type of albuminocytologic dissociation present in this and other cases reported may point toward some sort of radiculoneuronitis. At least it must be admitted that there is sufficient irritation of the central nervous system to produce pronounced elevation in protein content in the spinal fluid. Why, in the case herein reported, there should also have been a reversal of the albumin-globulin ratio is a mystery, unless one might postulate damage to the protein-forming function of the liver and reticuloendothelial system accompanying the diabetes of long standing.

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Ruptures of the Umbilical Cord with a Case of Intrapartum Rupture of All Three Vessels

H. E. Bowles, M.D., and R. D. McKee, M.D., *Honolulu*

FOR practical purposes it would seem fitting to classify ruptures of the umbilical cord as (a) partial, and (b) complete. Under partial ruptures should be included:

1. Rupture of a varix of the umbilical vein.
2. Tearing of one or two of the three blood vessels.
3. Discontinuity of the epithelium covering the cord, with resultant extrusion of Wharton's jelly.

Bleeding from vessels in velamentous cord insertion is not uncommon. Partial ruptures have been discussed by Forsell,[5] and cases reported by a number of authors. In a case reported by Page,[7] a bleeding placental artery was tied prior to delivery and the child was saved.

Complete ruptures of the cord have been reported by Dyrenfurth,[4] Douglas,[3] Siddall[10] (first case), Baldi,[1] McClintock,[6] Bloxsome,[2] and Sackett.[9] Although the authors could find only these reports of a total of seven cases of complete cord rupture in the literature available in Honolulu, no doubt it is not too rare an occurrence.

ETIOLOGY

Among the causes of cord rupture are: (a) velamentous cord insertions, (b) absolute or relatively short (by wrapping about neck or extremities of fetus) cords, (c) localized necrosis in the cord, (d) hemangioma of cord vessels, (e) trauma due to podalic version, forceps blades, etc., in manipulative deliveries.

From the Obstetrical Service of the Kapiolani Maternity Hospital, Honolulu, T. H.

ANATOMICAL CONSIDERATIONS

According to Spivack[11] there is some evidence that there are nerve fibers in the intra-abdominal portions of umbilical arteries. In addition, Spivack brings out the fact that the blood vessels of the umbilical cord differ anatomically from other vessels of similar caliber in the following ways: (1) The presence of folds and nodules of Hoboken in arteries and of semilunar folds in the vein, (2) the absence of true valves, (3) peculiar distribution of elastic tissue, (4) strongly developed arterial media whose powerful contractions are sometimes ascribed to the spiral or snail-like course of its muscle fibers, (5) absence of vasa vasorum and adventitia, (6) presence of a delicate connective-tissue stroma which contributes to the sponginess of the veins, (7) a nerve apparatus not yet positively demonstrated.

As a result of these various factors it is brought out that the umbilical arteries are almost impervious to the blood stream soon after birth. This seems to explain why Rachmanow,[8] in 1914, reported several thousand cases in which the umbilical cords were not tied and not a single newborn died from hemorrhage. These anatomical considerations will explain at least in part why at death there was seldom more than a small amount of intra-uterine bleeding despite severing of the umbilical arteries intrapartum. McClintock,[6] describing a case in which there was no bleeding from either end of the severed cord, concluded that the infant had died of asphyxia rather than hemorrhage. In several other cases also the amount of blood was minimal.

CASE REPORT

A 25-year-old primigravida was admitted to the Kapiolani Maternity Hospital Nov. 7, 1947. The estimated due date was Oct. 26, 1947. The course of the pregnancy had been uneventful prior to the onset of labor, and all laboratory tests revealed normal findings. Onset of labor was spontaneous, and the first stage was 15 hours and 20 minutes long. The second stage lasted 26 minutes. The fetal head engaged in the left occiput transverse position and apparently maintained this position until the cervix was fully dilated. Fetal heart tones had remained strong and regular until the fetal head reached to 2 cm. below the ischial spines. At this stage, the fetal heart rate rose suddenly and without warning to a rate too rapid to count and then suddenly ceased. No external bleeding was noted. The patient was draped quickly and under light cyclopropane anesthesia the fetal head was rotated easily from left occiput transverse to right occiput anterior position, using the Barton forceps. The delivery was completed by delivering the fetal head with Simpson forceps. Left mediolateral episiotomy was done. The fetal head was at plus 2 cm. below the ischial spines at the time of delivery and the cervix was fully dilated. Membranes were artificially ruptured just prior to the delivery. A large amount of meconium escaped along with amniotic fluid but a very small amount of dark red blood was present. The umbilical cord was looped once about the infant's neck and once in a complete turn around its right arm in the antecubital space. Where the cord crossed the antecubital space all three vessels—the two umbilical arteries and one umbilical vein—were completely severed. The cord was held together by a shred of epithelium not more than 0.5 cm. wide and infinitesimally thin. This was regarded as a complete severance of the cord inasmuch as the three vessels were totally cut through and the ends widely separated. There was no active bleeding in progress. The total amount of visible blood was not more than 5 cc. Trauma such as pinching of the cord by the forceps during delivery was ruled out by careful examination of the fetal head at the time of forceps application and prior to traction. A search was made for prolapsed cord and none was found.

Careful inspection revealed that the tear had occurred at a natural kink in the cord. This kink showed a lesser and a greater curvature and it would seem that the continued tension on the cord in late second-stage labor caused the shorter and inner portion of the kink in the cord to give way, shearing off the three umbilical vessels and leaving a narrow shred of the greater curve of the kink still holding.

It is believed that intra-uterine death occurred by asphyxia as it did in McClintock's[6] case, rather than by hemorrhage.

SUMMARY AND CONCLUSIONS

Partial severing of umbilical cord vessels has been reported fairly commonly in the medical literature.

We have been able to find reports of only seven cases of the severance of all three umbilical vessels during labor.

An eighth case of intrapartum avulsion of all three vessels is presented.

Etiology is discussed and attention is called to the role of actual and relative shortening of the cord in causing cord severance, especially if the cord contains kinks.

There is strong evidence that fetal death is more apt to occur from asphyxia than from hemorrhage, as blood loss is usually minimal when the vessels are completely severed.

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California Cancer Commission Studies*

Chapter XXIX

Tumors of the Brain and Spinal Cord

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IF a brain tumor is suspected, the diagnostic aids to prove or disprove its presence are numerous. Thirty years ago they were unknown. These are pneumoencephalography, electroencephalography, and arteriography. By one or the other, and not infrequently by information from all three, the site of the neoplasm can be determined, and the pathologic structure and size of the lesion as well. All of this has contributed to an early identification and given assurance in the selection of the surgical approach and the method of treatment.

The responsibility for suspecting a tumor in its early stages rests upon the physician who is consulted first. Under what conditions, then, should one suspect a neoplasm? Obviously not when one is confronted with fever and evidence of infection, and not when there are clear indications of more than one localization, such as signs pointing to both hemispheres or to several areas in the nervous system.

When the signs or symptoms can be explained on the basis of a single lesion *which is progressing*, one should think of tumor. The rate of onset gives a clue. An embolic lesion appears instantaneously, a hemorrhage occurs in a matter of minutes, a thrombosis usually over hours. Tumors give indications of their presence over weeks or months. A progressive single lesion implies growth, and adequate diagnostic methods are required. One should not delay for the appearance of signs and symptoms indicative of its space-consuming ("chorophagic") nature, such as choked disks, headaches, vomiting, or other evidences of advanced intracranial pressure. In the late stages, the individual harboring a tumor presents evidence of a single, progressive and chorophagic lesion.

The presenting symptoms may be varied. Contrary to the prevailing impression, chronic, constant headaches are not indicative of tumor. Adults who, without previous head injury or syphilis, develop *fits* are suspect. *Progressive paralysis, aphasia or dysphasia, complaints referable to ear or eye with increasing loss of function,* all demand investigation. Too often, in the absence of neurological findings, complaints combined with some *changes in temperament* cause the patient to be referred to the psychiatrist, and the true nature of the trouble is unrecognized. *Vomiting* as a primary symptom is not infrequently seen in children with posterior fossa tumors, and may antedate the appearance of other signs by weeks or months so that psychogenic cause may be suspected. In the cerebral tumors of adults, vomiting is not a prominent symptom.

The neurological findings on clinical examination, together with plain x-ray films, are usually sufficient to diagnose a tumor. The diagnostic aids employed by the neurological surgeon are required for more accurate localization so that the procedure selected for removal will involve the most direct approach with minimum tissue resection. They also give additional information useful in forecasting the type of tumor, the variety of neoplasm, or the presence of an aneurysm.

Some generalities may be permitted. Subtentorial tumors predominate in childhood; viz., the midline medulloblastomas originating in the roof of the fourth ventricle, the astrocytomas unilateral in origin, and the less frequent ependymomas. The craniopharyngiomas (Rathke pouch tumors) in most instances give evidence of their presence in childhood.

Supratentorial tumors predominate in adults, particularly in middle age, and are more common in the anterior and middle portions of the hemispheres than posteriorly. The origin of the meningiomas is indicated by their name. In this sense they are not truly brain tumors. They grow over a period of years, rarely become sarcomatous, but may invade or penetrate bone in advanced stages. Ordinarily they are encapsulated, benign, and do not recur if removed with the invaded dura. They are rare in the young and reach the peak of their frequency in adults around the age of 45. It is in persons in this age period, too, that the acoustic neurinomas are observed. These posterior fossa tumors progress over years, giving rise to tinnitus with progressive deafness and nystagmus long before intracranial pressure and cerebellar signs appear. In the cerebral hemispheres, chiefly in adults, are seen the slowly growing, relatively benign gliomas—the astrocytomas and oligodendrogliomas—which repay radical removal with long periods of life and even cure.

The *bête noir* of the brain surgeon is the glioblastoma multiforme. Highly malignant, rapid in growth, often involving motor or speech areas, occurring in the most active period of middle age, relatively insensitive to x-ray, it recurs within weeks or a few months after even the most radical removal.

There are four types of pituitary adenomas. The chromophobe adenomas, most common of all, occur in middle age, and failure of vision or field defects bring them to the attention of the physician. It may then develop that the patient, if a woman, has con-

*Organized by the Editorial Committee of the California Cancer Commission.

sulted the gynecologist because of amenorrhea, or if a man, because of impotence. The eosinophilic adenomas are usually recognized because of the action of the acidophilic cells, which secrete growth hormone and produce gigantism or acromegaly or both. The basophilic adenomas are suspect in "Cushing's disease" but they present clinical pictures indistinguishable from certain primary adrenal growths. The malignant pituitary adenomas are so called largely because they have burst their capsular confines.

Metastatic tumors to the brain from distant primary sources constitute perhaps 3 per cent of brain tumors, and frequently attract attention before the primary tumor is evident. The lung, breast, and melanotic growths are likely sites of origin. The bowel and adrenal gland are less commonly the sites of origin.

Primary intracranial tumors have a distinctive way of spreading, not through the blood stream or lymphatics, but by seeding and sowing implants along the pathways of the cerebrospinal fluid. By this process it is common in the medulloblastomas of children to have the late development of a paraplegia from a separate implant in the spinal canal. Such a spread is less often seen with glioblastomas and ependymomas, and rarely with astrocytomas. In countries where echinococcus infestation is common, multiple space-consuming lesions have this special implication.

By roentgenographic study, even without the special techniques of air injection or arteriography, additional diagnostic information is readily obtained. The films may show the results of increased pressure; namely, the separated sutures (readily anticipated by a cracked-pot note on percussion of the skull), or the beaten silver appearance. The former occurs in younger children and the latter seldom is seen in patients beyond the middle of the third decade. The pineal, if calcified, may be shifted away from the hemisphere of greater volume and higher pressure. An enlarged sella turcica may indicate a hypophyseal adenoma, a flattened or enlarged one point to a Rathke pouch tumor with its spotty calcification. A spread and enlarged sella should always, however, bring to mind the possibility that one is dealing with a large third ventricle with hydrocephalus. Localized bone changes — thickening, rarefaction and destruction—are all seen at various stages with meningiomas. Metastatic tumors, epidermoids, and myelomas are accompanied by fairly characteristic bone changes. Certain tumors are prone to show calcifications. The Rathke pouch tumors have been mentioned as one variety, but a high percentage of the oligodendrogliomas do also, and a considerable number of astrocytomas and aneurysms. In other lesions calcification is less frequent.

Successful surgical treatment of intracranial tumors is dependent upon their accessibility, their type, and the stage at which they are diagnosed. The outcome is determined by the extent of involvement of important areas (motor, speech, etc.). Approximately 55 per cent of all intracranial tumors fall within the favorable, i.e., removable group, granting that a reasonably early diagnosis is made and that surgical treatment is properly executed. Improved anesthesia, the electrosurgical unit, the newer hemostatics, and the antibiotics, have supplemented the diagnostic aids mentioned above. As a result, tumors formerly unapproachable, or removable only in part, can now be eradicated. With these advances also, the operative mortality for intracranial new growths varies between 10 and 15 per cent.

Neoplasms of the Spinal Cord

Twenty-five years ago a survey of the cord tumors treated at the University of California Hospital revealed that each patient consulted on an average of 18 physicians before a diagnosis was made. Today the situation is vastly different, and the diagnosis is usually suspected before an advanced stage of paralysis is reached.

The *bilaterality of sensory, motor or reflex changes*, in the lower extremities alone, or in all four extremities, indicates the likelihood that the site of trouble is in the spinal canal. The progressive feature suggests a growth. A partial or complete blockage of the canal shown by spinal puncture and the Queckenstedt test confirms the physical characteristic of the lesion.

A constant radiation of radicular pain, usually worse at night, may indicate tumor long before other signs appear. Such a complaint merits a spinal puncture, Queckenstedt test, estimation of the spinal fluid proteins, and x-ray study. With the establishment of a definite level of sensory alteration and gradually developing disability below it, a growth should be diagnosed.

Fifty per cent of primary cord tumors are encapsulated, benign meningeal tumors or neurofibromas. The latter may be multiple in von Recklinghausen's disease. With such extramedullary tumors the sensory changes, although not necessarily uniform, are more pronounced in the distal parts of the extremities. Involvement of the sphincters is a late manifestation. Pain, not usually intense, is greater than with the intramedullary growths. The latter are apt to be ependymomas, other types of gliomas, or epidermoids. Sensory changes may, with laterally placed tumors, be of Brown-Sequard type. The intramedullary tumors produce the most marked sensory changes in the segments corresponding to their extent in the cord, rather than in the distal parts of the extremities. Tumors in the lumbar canal, because of involvement of the cauda equina rather than of the spinal cord, are associated with decreased reflexes in contrast to the increased reflexes characteristic of cord lesions. They should always be suspected when bilateral sciatica is present. These tumors, because of the ready displacement of the cauda, may attain huge size before neurological signs appear.

Variations in the neurological picture caused by cord compression are as numerous as the possible locations of the lesion from sacrum to foramen magnum. When compression of the cord is suspected, spinal puncture and the studies mentioned above should be performed. When further details of the level of the lesion are required, fluoroscopic study with x-ray pictures may be obtained by the introduction of radiopaque material into the spinal canal. Pantopaque is at present the most satisfactory material used for this purpose and may be aspirated and removed under the fluoroscope at the conclusion of the study.

Bone disease which causes cord compression may be demonstrated by plain x-ray films. Primary bone growths, such as giant cell tumors, and metastatic lesions, from the prostate or other sources, are not uncommon. The granulomas, particularly tuberculomas and their associated extradural abscesses, may be shown. Because of the common extension of extradural tumors through the intervertebral foramina, giving rise to the shape known as dumb-bell or hour-glass, complete x-ray studies should include three-quarter views as well as anteroposterior and lateral pictures. These may show enlarged foramina. Widening of the spinal canal by enlarging tumors may be demonstrated.

The extradural tumors include growths of blood vessel origin, occasional lipomas, chondromas, and some of neurogenic origin. These are frequently of the hour-glass type.

Surgical removal of the benign, intradural but extramedullary tumors, which constitute about half of all cord tumors, is highly satisfactory, the results being brilliant and the operative mortality about 1 per cent. Except in neglected cases, complete recovery is obtained. With the other types, operative removal and relief of cord compression may be combined with deep therapy.

SUMMARY

One should think of a tumor of the central nervous system when the signs and symptoms can be explained on the basis of a single lesion which is progressing.

The diagnosis of a tumor is established if in addition there are evidences that the lesion is space-consuming. These evidences are signs of increased intracranial pressure with brain tumors and indications of blockage of the spinal canal as shown by the Queckenstedt test with cord tumors.

"Bone Tumors" by Don King, M.D., Chapter XXX of the California Cancer Commission Studies, will be published in this section of the June issue of CALIFORNIA MEDICINE.

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EDITORIALS

Industrial Cases

California doctors are continuing their effort to secure the adoption or recognition of a schedule of fees for industrial cases which shall be fair, reasonable and equitable. Such a schedule has not heretofore existed and it seems that the path toward securing one is beset with numerous obstacles. Nevertheless, the effort shall and must continue.

Last September the California Medical Association presented its application to the Industrial Accident Commission for approval of a proposed schedule of fees for compensation cases. The Commission in November tabled this application and, at the same time, disowned any statutory authority to make and enforce a schedule of medical and surgical fees. Simultaneously, the Commission announced its intention of recognizing until next June 30—as it has done since 1914—a schedule of fees previously adopted officially by itself. Thus, in one breath, the Commission blows hot and cold on its own authority to set compensation medical fees. Both doctors and insurance carriers have been marvelling at this thermal feat.

The California Medical Association then came out with the recommendation that its members adopt for their own use the fees recommended in the September 1947 petition to the Industrial Accident Commission. Many of the insurance carriers countered by agreeing among themselves to pay only those fees adopted as an official act of the Commission in 1946. Today, under this situation, the doctors are charging one set of fees and some insurance carriers are paying another. To the credit of many insurance carriers and self-insurers, it should be recognized that the proposed 1949 fees are being paid without question in many cases.

A possible end to the impasse is being tried out in the meeting of joint committees of the Association and the insurance carriers. These committees are engaged in the effort to adjust differences of opinion and to arrive at a schedule of fees which will be mutually acceptable to physicians and insurance interests. Such a schedule is distinctly within the realm of reason, provided only that reason prevails in discussion of it and in the attitudes of the participating conferees. The Association's committee has been selected on the basis of experience and reason alike, and the insurance representatives have been chosen on a similar basis. Thus there appears every chance for a successful conclusion to this perennially difficult problem.

The principal stumbling block confronting the joint committees are the time limit of next June 30 (after which time the Industrial Accident Commission has decided to wash its hands of the entire matter of fee schedules) and the presence before the State Legislature of several bills which would grant specific statutory authority to the Commission to promulgate and enforce schedules of fees and charges for medical, hospital and other services provided to injured workmen.

If the June 30 deadline is passed, there is bound to be tremendous confusion in the whole field of industrial medicine. Doctors will need a guide in assessing their fees for services rendered and insurance companies will no doubt expand their efforts to establish limited panels of physicians to treat injured workmen, probably at reduced fees. If the bills now up in Sacramento are adopted, the Commission will be given absolute authority to set medical and surgical fees at any level and possibly at levels which

have no bearing on the actual value of such services. This blanket authority does not appeal too much to those providing the services, which would be unlimited in the scientific sense but under strict regulation and limitation in the economic way.

The California Medical Association is geared to meet whatever developments take place in this matter of industrial fees. Its obligation is to its entire membership and not primarily to those few physicians who see fit to contract with insurance carriers for medical services under conditions and at rates which are impossible, economically and ethically, for their many colleagues.

It is to be hoped that the dangers inherent in the present and proposed programs of departmental control over professional practices may be avoided by common-sense activities by the joint medical-insurance committees now meeting. The solution lies in the recognition of the respective rights of the parties at interest, primarily those of the injured workman.

Letters to the Editor . . .

Gluten Intoxication

It was shown by Wagner and Elvehjem[3] that "running fits" or so-called "canine hysteria" can be induced in dogs by feeding them a ration containing wheat gluten. Convulsive seizures typical of these attacks suggested the possibility that canine gluten toxemia might be related to human epilepsy.[1] To test this possibility, Newell[2] and his associates of the Departments of Neurosurgery and Medical Electronics, University of Wisconsin, determined the electroencephalogram changes induced in dogs by gluten feeding and compared them with the known changes observed in epileptic patients.

Eighteen mongrel puppies ranging in age from six to eight weeks were used in this work. Each animal was dewormed and dusted with D.D.T. before the experiment was begun. For a week or more before the operation and during the period when normal electroencephalograms were taken, each animal was maintained on a control diet containing 20 per cent casein as its main source of protein.

With the animals under deep nembutal anesthesia, four tantalum electrodes were placed extradurally in each dog. The technique was the same as that used in clinical electroencephalography except that the electrodes were anchored firmly to the bone in order to minimize movements. All records were taken without the use of any anesthetic or drug. After several satisfactory normal records were obtained, 20 per cent wheat gluten was substituted for casein in the control diet. Generalized convulsions were frequently observed within three to ten days after this change, although an occasional dog was fed gluten for as long as five weeks without an epileptic seizure.

In a typical record on the control diet the electroencephalogram (E.E.G.) showed a moderate voltage activity with a predominant frequency of 8 to 10 per second. On changing to a diet including wheat gluten no change in voltage activity was noted during the first eight days. On the 11th day there occurred a definite change in the E.E.G., with the appearance of high voltage slow waves at a frequency of 2 per second. No outward change in the appearance of the dog was noted until the 21st day, when a series of convulsions occurred. A record taken two minutes after one of these convulsions showed many spikes with some low voltage waves at a frequency of 5 per second. A record two days later showed occasional high voltage waves at a frequency of 2 per second. Both the seizure and interval E.E.G. were thus comparable to those recorded in human epilepsy.

The following day casein was substituted for gluten in the diet of this dog. Seven weeks later the E.E.G. showed a normal pattern with a moderate voltage activity with 8 to 10 waves per second. One month later there was a temporary partial reversion to the wave pattern seen when the animal was fed gluten. This fluctuating abnormality in E.E.G. after gluten feeding has been observed on several other dogs.

This apparent identity of human epilepsy and canine gluten convulsions suggests that a more detailed study of this parallelism may lead to conclusions of practical clinical interest. Such detailed work is now in progress in the Wisconsin laboratories.

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NOTICES AND REPORTS

The A.M.A. Plan of Campaign Against Compulsory Health Insurance

STRATEGY AND POLICIES

Presented by Clem Whitaker and Leone Baxter, Directors of the National Education Campaign of the American Medical Association, for the Conference of State Medical Societies, Chicago, February 12, 1949.

CLEM WHITAKER: American medicine, in its campaign against compulsory health insurance, cannot afford to fight alone.

This must be a campaign to arouse and alert the American people in every walk of life, until it generates a great public crusade and a fundamental fight for freedom.

We must make every American know that medicine is not simply fighting for self-interest, but is actually fighting to avert the creeping paralysis of bureaucratic regimentation of the people's lives.

We need the help of every American who honestly believes in the American way of life—and our campaign must be geared to that help.

Any other plan of action, in view of the drift toward socialization and despotism all over the world, would invite disaster.

Doctors don't need to stand alone in this battle. They must not stand alone. And it's our job to see that they don't stand alone.

That's the reason that the American Medical Association, in establishing its National Education Campaign, has set as one of its major objectives—the mobilization of other great national organizations representing other professions, trades and businesses; civic and fraternal, religious and patriotic groups; women's clubs, farm and veterans' organizations and every other association which is willing to make common cause with medicine in this battle.

That's one of the top priority jobs we are going to be organizing and directing from the national campaign headquarters. But the success of that drive, in the final analysis, will depend on whether it reaches below the national level—down to the grass roots, in every state and county and city in America.

Let's set the record straight, too, on the subject of lobbying—and the smear attack that has been launched against the A.M.A. in that connection.

The charge has been made by the advocates of compulsory health insurance that the American Medical Association plans to invade Washington with a high-powered lobby and a $3,000,000 "slush fund" in an effort to block passage of the legislation in Congress.

That charge is absolutely false—and every state and county medical society, and every doctor who values the good name of medicine, should see to it that this smear attack is branded as false in every community in America.

The Washington office of the A.M.A. is one of the most modest legislative offices maintained by any of the national associations in the capital—and is staffed by men of unquestioned integrity who are highly respected in Congress.

The American Medical Association isn't embarking on any high-pressure lobbying campaign in Washington, nor is there any "slush fund." The socializers in the Office of Social Security, who have used their government facilities and government funds to lobby Congress, are simply sending up a smoke-screen against A.M.A. as a cover for their own highly questionable operations, and the people need to be told that.

The A.M.A., in its campaign, is carrying its case to the people of America in a grass roots crusade which we hope, with your help, and the help of tens of thousands of others, will reach every corner of this country.

One of the greatest rights which we have as a free people is the right of petition—and we intend to exercise that right, even though Oscar Ewing and his socializing satellites will do everything in their power to keep us from getting our story to the people. If that is lobbying, it is lobbying in the finest American tradition. The American people, not Congress, will decide this issue in the final showdown, and Mr. Ewing knows that. That's why he fears the National Education Campaign which the A.M.A. has authorized—and that's why he will do his utmost to discredit and block us.

There's another important financial policy with regard to this campaign which you should know, too—and which should be made known to every state and county medical society, as it will help to keep the record straight and let doctors know how their funds are being handled.

That policy is this:

Every dollar expended by the national campaign office will be reported in a check-by-check accounting to A.M.A. And our financial reports will be available for government inspection at any time!

That is the complete answer to the insinuations and criticisms of medicine's enemies, or political demagogues who hope to make a whipping boy of the medical profession.

TWO MAJOR OBJECTIVES

Let's consider some of the other basic policies of the campaign.

The Coordinating Committee, in approving a broad, public campaign, beamed directly to the people, rather than just to Congress, established two major objectives:

The immediate objective is the defeat of the compulsory health insurance program in Congress—and there is great urgency in that phase of the problem. The first showdown battles on compulsory health insurance may come within the next 60 or 90 days. We can't afford to be lulled into over-confidence by reports out of Washington that no serious effort will be made to push through the program at this session of Congress. That may be propaganda emanating directly from the camp of our opponents.

The long-term objective is to put a permanent stop to the agitation for compulsory health insurance—and the most vital step in achieving that objective will be an all-out campaign to enroll the American people in voluntary health insurance systems. The A.M.A.'s Coordinating Committee has authorized and directed us to conduct a nation-wide educational drive to make America health-insurance conscious—and to work with the prepaid hospital plans, the prepaid medical plans, the accident and health insurance companies and all other sound groups in the voluntary field to achieve this objective.

This is an affirmative campaign, not just a negative campaign—and I think we need to hammer home that basic point in every public appearance.

We're not just working to beat a bill. We're going to work together to resolve a problem. We're going to do something about taking the economic shock out of illness!

That's the kind of program your A.M.A. is backing—and that's the only kind of program that will eventually and finally lay the ghost of compulsory health insurance in this country.

We have already held preliminary meetings with representatives of the medical care plans and the private insurance companies to pledge them A.M.A.'s vigorous assistance in promoting voluntary health insurance—and to enlist their aid, if possible, in stepped-up selling and advertising campaigns.

I believe there are representatives in attendance here today from the medical and hospital plans and the health and accident underwriters.

In the presence of the Board of Trustees and the Coordinating Committee of the American Medical Association, I want to make this unqualified statement concerning the A.M.A.'s National Education Campaign:

The accent in this campaign is going to be on the positive. We are going to do everything in our power to acquaint the American people with the desirability and the availability of prepaid, budget-basis medical care. We believe in voluntary health insurance, not just as a political expedient, but as a sound development in medical economics. We want everybody in the health insurance field to sell insurance during the next two years as he has never sold it before—knowing that he has the prestige of the American Medical Association, and all its power and facilities, squarely behind him. And we are going to ask the doctors, when they are talking to patients who are in need of budget-basis medicine, to take time to encourage them to enroll in a good, sound voluntary health system.

That's a fundamental part of the A.M.A. program—and I hope every representative of a state society in this room will take that message home with him.

FACTS AGAINST ARTIFICE

We have an inspiring case to present in our affirmative campaign.

More than 52 million Americans already have decided that the voluntary way is the American way to cope with this problem—and have enrolled in voluntary health insurance systems.

The tremendous growth of the voluntary systems has come in a very short span of years—and has been one of the most spectacular economic developments in our time.

If there was even half the demand for compulsory health insurance in this country that exists for voluntary health insurance, Oscar Ewing and the socializers would be on the road to victory and nothing could stop them.

But the major demand for compulsory health insurance in the United States is an artificial, trumped-up demand, generated by social bureaucrats. They are leaning heavily on the false lure of something-for-nothing, and the people's fear of the cost of unexpected illness, in a desperate drive to

drum up public favor for their political patent medicine.

On the other hand, American medicine and the voluntary health insurance systems already are near the half-way mark in the campaign to provide prepaid, budget-basis medicine for the American people within the normal framework of our free enterprise system.

The job is half done, but it is of urgent importance that it be completed. Every American who needs prepaid medical and hospital care should know that it is available to him. We need to take health insurance out of the luxury category—and let the people know that it can be bought economically and should be included as a necessity in the home budget, just as food and shelter and life insurance are budgeted.

The finest antidote for compulsory health insurance is voluntary health insurance—and the agitation for socialization of the medical profession will come to a halt when the majority of the people have been provided with the remedy.

That's the way we can accomplish our long-term objective—and every state and county medical society in the United States ought to become a strong ally of the insurance industry and the prepayment systems, and work with them until the final objective is achieved.

Now let's take a good, hard look at our immediate objective—the defeat of the compulsory health insurance program in Congress.

The time schedule of the opening battle in Congress we can't determine. Our opponents have that advantage—and our only safe course, regardless of the conflicting and confusing reports which will emanate from Washington, is to mobilize for all-out action now and be ready whenever the attack comes.

Our own militancy and our own readiness for a showdown may make the socializers hesitate to force the issue at this session of Congress—and may give us badly-needed time to get our long-range campaign under full steam. But we can't have any assurance of that desired development.

The fate of other bitterly controversial issues now pending in Congress may be a decisive factor in determining the tactics of our opponents. If other legislative proposals in the controlled-economy program of the Truman administration should be jammed through this Congress fairly early, we would be in real danger that the steam-roller would keep right on rolling—and in that event medicine might be engaged in a bitter battle for survival before the end of this session.

On the other hand, if Congress becomes embroiled in heated and long-drawn controversy on other issues which are ahead of compulsory health insurance on the agenda, we may have a breathing spell. Or we may find Oscar Ewing proposing a watered-down bill, hoping to disarm us with apparent moderation and get half a loaf this session with the full expectation that he will be able to get over the rest of his program later.

We can't call the shots on just how or when the battle will be joined, but we can and must get American medicine off the defensive and into an affirmative, offensive position. That's an immediate and vital necessity.

TRUTHFUL, HARD-HITTING CAMPAIGN

We have emphasized that this is going to be a sound, constructive campaign.

But let me underscore this statement:

This isn't going to be any panty-waist campaign!

The A.M.A. is going to wage a truthful, hard-hitting campaign, in language that the American people understand.

The critics of the medical profession have had their field day—and they'll continue to have it until American medicine strikes back and strikes hard.

There are going to be no punches pulled in our national publicity campaign—and we want you to know that.

We're going to attack—and attack—and attack—until the truth about the vicious consequences of political medicine are known throughout this country.

We're going to put the foes of American medicine on trial before the bar of public opinion in this nation—and let the people decide for themselves whether they want men of medicine, or medicine men, in charge of the health of their families.

We're going to expose the shameful misrepresentation, the juggled facts and garbled statistics, the phony draft rejection figures and the deliberate attempt of Patent Medicine Man Oscar Ewing to hide from the people the true cost and the social consequences of the scheme of socialized medicine which he is proposing.

That's one of the jobs we have at the head of the list in national headquarters—and we hope that all of you, in your home states, will duplicate it.

If we're going to turn the tables on the socializers, and get the medical profession into an affirmative position, there's another immediate job to be done. That's the job of mobilizing organization support for medicine's cause in agriculture, business, in industry, in the veterans' organizations, in the women's clubs, in churches and lodges—and in all the thousands of organizations which make up a cross-section of America.

The fastest way to make our influence felt in Washington is to marshal a powerful array of nation-wide organizations, representing great groups of American citizens, in opposition to compulsory health insurance.

That job is the direct responsibility of our national headquarters and the drive for specific action by hundreds of national organizations, trades and professions already has started. But we'll need lots of help from all of you in making this phase of the campaign successful.

In moving for important endorsements of medicine's position, medical leaders who have the contacts often will be called on to help open the door or close the sale.

From these major endorsements will stem much of our publicity in the early days of the campaign,

designed to broaden the campaign into a great public crusade. We need allies, strong allies, whom we can convince that this is their battle just as much as ours. We need the use of their mailing facilities, space in their newsletters, house organs and magazines. We need letters and phone calls and telegrams from their members, flowing to their congressmen.

All of this requires manpower. Our small professional staff can't possibly do all the work, but we believe that we can find doctors and others who will do it. Mainly, this is the same task which confronts a military organization. The troops fight the battle, but they first have to be properly mobilized, trained and directed.

STATE AND COUNTY ACTION NEEDED

In every state and in every county there should be similar organization drives—for action by state and local organizations which help to mould public opinion. We need the impact of their support on your congressmen—and your United States senators. We want them to hear from the organizations at home, so that they know how their own constituents feel on this issue.

One of the objectives of the national headquarters will be to provide you with all the materials of war—and to lighten the financial load on the state and county societies. We will attempt to provide you with pamphlets and posters, form speeches, cartoons, mats and other supplies in any quantity you can put to good use. We hope the only limit that will be placed on the materials available to you will be the limit of your ability to get them into the hands of voters in your home states and communities.

The question has been asked whether the A.M.A. will also allocate funds to the states to help in distributing costs, or for other purposes. The answer to that question is "No," for reasons which probably require no explanation.

I want to touch briefly on another probable development in the national campaign which is still in the evolutionary process, but which you will hear about in more detail within the near future.

We have recommended that a national committee of leading citizens in all walks of life be established as auspex for an important part of the work in this campaign—a committee which we will help to organize, but which will also draw strength from many other sources.

This organization will be called the American Committee for Health Security.

Under the committee's name, on one side of the letterhead, will be the slogan: "For Voluntary Health Insurance; Against Compulsion!" On the other side, balancing this, will be a second slogan: "The Voluntary Way Is the American Way."

Some of the great, outstanding leaders of America have agreed to serve as members of this national lay committee—and we believe it will broaden our front and create a rallying place for thousands of people who have no direct connection with medicine, but who have a healthy interest in the welfare of America.

When the time comes for perfecting that organization, we may ask each of you to help recruit some of the outstanding lay leaders in your state for that committee.

PRACTICING PHYSICIANS MUST HELP

Now let's return to the most important man in this campaign—the practicing physician!

The 150,000 members of the American Medical Association must be the front line troops in this battle.

The A.M.A. and the state and county medical societies can't win this fight, but their members can.

A doctor can talk to his patients on this issue and get their earnest attention, because this is an issue that involves their health and their relationship with the doctor.

Our greatest need—and this is the most important job you will have—is to get the word to every doctor that this is an emergency, that his help is needed, and that his right to continue in private practice may depend on how he measures up to the challenge.

We need every doctor on fire on this issue . . . taking time out to talk to every leader he knows in the community, urging them to write their congressmen, stirring his patients and friends into action.

A doctor knows that political medicine is bad medicine—that it means hit-or-miss diagnosis and superficial treatment of symptoms. He knows that personal interest in the welfare of the patient suffers when assembly-line medical practice takes over. And he knows that the quality of medical care steadily deteriorates when doctors succeed or fail on the basis of political preferment, or on the number of cases they can rush through their offices in a crowded day.

No one can talk to a patient on that subject with the eloquence of his family doctor—and we simply must have that doctor at work, if this battle is to be won.

Doctors know, too, if they are familiar with what's happened in other countries, that invasion of the individual's privacy is one of the most objectionable features of compulsory health insurance. They know that the sanctity of the physician-patient relationship goes out the window when government medicine comes in.

That's a subject on which a doctor can talk convincingly—and our campaign pamphlets will provide corroborative material on that personal, compelling issue.

People talk to doctors about their financial troubles as well as their physical ills—and they'll listen to the doctor if he tells them that compulsory health insurance isn't free—that, instead, it will mean a 6 or 8 per cent payroll tax on every dollar they earn.

That's the kind of missionary work that will win this campaign—and that, more than all else, will give us a real grass roots campaign.

How the Doctor's $25 Will Be Spent

LEONE BAXTER: Every minister preaches from a text—and every campaign, if it is a successful campaign, has to have a theme.

The theme, if it is geared to reach more than 100 million people, as we must in this campaign, should have simplicity and clarity.

Most of all, it must high-point the major issues of the campaign with great brevity—in language that paints a picture understandable to people in all circumstances.

EVERY DOCTOR A CAMPAIGNER

That's one of the reasons we have a large, blown-up color reproduction of the famous Fildes painting, "The Doctor," on exhibit here today, with the simple caption under it: "KEEP POLITICS OUT OF THIS PICTURE!"

The picture and the caption, even without elaboration, focus attention on one of the most important arguments against government-controlled medicine.

Smaller color reproductions of this famous painting soon will go up in doctors' offices all over America as one of the first steps in dramatizing our case to the American people—and, more important—as the first step in making doctors campaigners in their own behalf. For this purpose we have added a hundred words of text which help to establish the theme of this campaign.

I'm going to read you that text, because it stresses, in simple language, the essential points of the case which we believe will turn the tide against compulsion and in favor of voluntary health insurance.

The text is as follows:

KEEP POLITICS OUT OF THIS PICTURE!

When the life—or health—of a loved one is at stake, hope lies in the devoted service of your doctor.

Would you change this picture?

Compulsory health insurance is political medicine. It would bring a third party—a politician—between you and your doctor. It would bind up your family's health in red tape. It would result in heavy payroll taxes—and inferior medical care for you and your family. Don't let that happen here!

You have a right to prepaid medical care — of your own choice. Ask your doctor, or your insurance man, about budget-basis health protection.

This is signed: American Medical Association.

These smaller posters will be sent under the signature of the A.M.A. to show medical men throughout the country that the Association is resolutely behind the national campaign. They will be sent to doctors only at their own request. Return postal cards will be in the mail shortly.

The dimensions of the posters are approximately 18 by 20 inches. They are dignified—but carry a strong message—stronger, we are aware, than most doctors are accustomed to display in their waiting rooms. Their final cost, including the right to reprint the famous picture, art work, stock, printing and mailing comes to about 30 cents each. If we can light the crusading fires, and tie into the campaign the majority of the doctors of this country, for the cost of 30 cents each, the results will be well worth the price!

THE REAL AMMUNITION

The major portion of the campaign budget will be spent for production of materials—the campaign ammunition. We are not going to waste any campaign funds on faulty ammunition. Any general pamphlet produced will have to be printed in minimum lots of 7,500,000—just to put 50 copies into each doctor's hands alone. To make the smallest trickle beyond that outlet to the public, we shall have to print a minimum of 10 million copies of any piece produced. That means simply that we can't afford to experiment. We can't afford to throw our next-best or divided efforts into print and hope it will suffice. What we produce must be brief enough to read—dramatic enough to create sentiment—and sound enough to produce action from the thinking people of this nation.

Some very excellent basic material had been produced by men of medicine and men close to the profession, long before the National Education Campaign was initiated — and that will give the production of the new material the most helpful impetus.

One of the pamphlets in the planning and production stage is a small, sparked-up human-interest folder to satisfy the need among doctors for a simple piece that can be given to patients, mailed with statements or placed in waiting rooms. It will be suitable as well for general use by allied professions and industries. This will be a special appeal, illustrated public pamphlet, geared to the interests of the average citizen—the veteran, the farmer, mother, businessman, wage-earner, etc.

The doctors will receive their first copy of that pamphlet direct from A.M.A. headquarters, with a brief letter, telling some of the highlights of the proposed campaign, and advising the doctors that they can get the pamphlet in quantity through their state or county medical societies.

The question-and-answer pamphlet which actually will serve as the doctor's campaign handbook, also is in process. The handbook should give every doctor not only the facts he needs to argue his case effectively, but also simple instructions on how to practice on the body politic.

The small leaflet will be a general public piece and can be distributed through many channels. The handbook, while beamed to doctors, also will be used for distribution among members of our lay committees. We believe that the dentists' associations, the druggists' organizations, the hospital associations, the medical auxiliaries and various other closely related groups should be urged to turn out similar material, or use ours under their own imprints.

We also plan to assist many cooperating national organizations—veterans' groups, for example—to produce pamphlets slanted to their own memberships, and emphasizing the arguments which will have special appeal to them.

A third pamphlet is in preparation whose title will be "Calling Every Doctor—This Is An Emergency!" This, too, will go directly to physicians, with a letter from the American Medical Association. It will be a briefly presented statement of the issue, the objectives—and the procedure to accomplish those objectives. Its purpose will be similar to that of the poster—to get every doctor who believes in the private practice of medicine working enthusiastically with his local campaign committee.

A PAMPHLET CAMPAIGN

Actually, this issue is made to order for pamphlet presentation . . . and we plan heavy use of pamphlets, running into many millions of copies, to tell medicine's story dramatically and effectively both to leaders of public opinion and to rank and file citizens throughout the country.

With new developments and changing conditions in the campaign, there likely will be need for frequent revision of the text of early pamphlets, or the production of entirely new material. As a consequence, a heavy load will fall on our writing and production department, and one of the first problems, of course, is to produce copy fast enough to satisfy the press, A.M.A. members, state and county societies and the literally hundreds of business and civic groups which all require special servicing.

Above all, the written material in this campaign must be emotional, fighting prose. We can't win an audience with dry, statistical copy. We have to give the people facts, but in very readable form. The surest way to break down apathy and public disinterest is to turn out copy that stirs the emotions—and, in doing so, opens closed minds.

It is vital, too, that much of this flow of words should reach the people through normal newspaper and magazine channels, rather than through direct publicity releases. We intend to work with the great newspapers and the national magazines to get them to do special jobs, with real reader interest, and that work already is well started. The story of British medical practice today, as an example, is actually one of the most important stories of this era. The A.M.A. already had started the wheels moving to get the truth and publicize it before this campaign began. It is a story which must be told and re-told by staff writers of American magazines and newspapers.

Once it has been printed in a magazine or newspaper of national importance, reprints of the article will be placed in the hands of key people throughout the country.

ORGANIZATION PHASES

There are two distinct phases of organization activity planned.

First is the plan of organization and operation for medical groups, which involves the relationship of the state and county societies to the A.M.A. in the conduct of the campaign. This calls for a definite division of work, with fixed responsibility in each area, so that a vigorous grass roots campaign can be developed.

Second is the plan for mobilizing the strength of the major public organizations, local, state and national—groups like the farm organizations, the more powerful business and civic associations, fraternal, religious and veterans' organizations.

The program with respect to the medical organization structure has been discussed carefully with the Campaign Coordinating Committee members in order to reach practical and intelligent decisions. The job must be done with as little friction as possible, so that doctors in the field will be directing their energies to winning converts, and their fire to the opposition. It is usually difficult to get 150,000 individualists (and most doctors are individualists) to agree on anything, but if ever the members of the medical profession needed to pull together, this is the time.

STATE MEETINGS

A number of states have reported that within the next 30 to 60 days after the National Meeting of State Representatives (February 12, in Chicago), they are calling meetings of county society representatives in their own states, for the purpose of passing along to them the national plan of campaign, and determining on their own procedures in relation to it.

Accordingly, for their help and guidance at this time, we will chart some of the important work which needs to be done within the states.

COUNTY SOCIETY ACTION

1. Every county medical society in the United States should adopt a strong resolution against compulsory health insurance within the next 60 days—and should then direct the president of the society to communicate its action, by letter or telegram, to the congressman (or congressmen) representing the district; also to the state's two United States senators. The society's resolution should emphasize the inevitable deterioration of medical care and the danger to the public health, once government medicine is in operation, and should stress the tremendous growth of the voluntary systems and that the American people are taking care of the problem in the American way. The president's letter to the congressman or senator should ask for a reply, so that the legislator's position can be made known to the doctors of his district. Copies of all replies should be forwarded to the national campaign offices and to the Washington office of A.M.A. as rapidly as they are received. We will provide several form resolutions as a guide to the societies.

The combined political strength of all the doctors in a congressional district is impressive—and we need to put every congressman on notice of the position taken by his doctor constituents.

LAWMAKERS' DOCTORS

2. We need to locate the personal physician of every congressman and every United States senator (the county society secretary probably should take on that job) and have him send a personal letter to his patient, the congressman, telling him of the danger of socialized medicine, and asking for his help in defeating any compulsory health insurance program which may be submitted. We will provide form letters, but the society secretary should help the doctor, if necessary, in rewriting and personalizing the letter in each instance. This letter also should ask for a reply—and again, the information in the replies should be sent to the National Campaign office and the A.M.A. Washington office.

SPEAKERS' BUREAUS

3. We need an active speakers' committee in every county society to cover local meetings. The executive secretary of each of the state societies should aid in organizing this work. We will provide form speeches, but in many cases they will need to be localized to meet local conditions.

One of the very great requirements is for every state to develop top bracket speakers, both in the profession and apart from it, who can be called on for important meetings, both state and national.

DEBATES

We do not believe it a sound campaign practice to sponsor too many debates. They make a forum for the opposition which would be difficult for them to secure otherwise, and they are too easily stacked. This is particularly true of broadcasts of debates open to the public. Our speakers will stick to the facts. But already in this campaign, the opposition has begun to use the facts very loosely. Their claques in the audiences are briefed to applaud wildly every trick phrase their speaker utters. And the public has no way of knowing which is fact and which is fancy. If our case were so poor that we had to stack meetings, it would not be worth the effort we shall all put into this campaign.

PRESS COMMITTEE

4. We need a press committee in every county society to make personal calls on the editors of all newspapers in the county and urge their support of medicine's position. This work, again, should be coordinated by the state society.

ENDORSEMENT DRIVE

5. Since our first objective is making the position of the people on this issue known and recognized by our representatives in Congress, much of our first campaign effort must continue to be devoted to getting organizations on record in opposition to compulsory health insurance.

In this connection, one of the first mailings from the national campaign headquarters to the states will be a list of conventions scheduled in each state during 1949. This is not to be considered a complete list, but we sincerely hope it will be helpful in beginning the drive for resolutions in your area. It will include all conventions reported at this early time; you will need to add to it as others are scheduled. The list will contain the following information:

Name of organization
Town where convention is scheduled
Estimated attendance
Person to contact, to our best knowledge
Whether convention is national, state or local.

Some organizations on the list may have a policy of not taking action on public issues, or will profess "no interest." When an issue is of fundamental importance, as ours is, however, and when somebody takes the time to explain the honest facts and drive for a resolution, virtually all the important organizations do take action.

As fast as they are produced, form speeches geared to different types of audiences will come along to you; also suggested form resolutions which can be localized or particularized as you see fit; also the question-and-answer pamphlet providing brief, factual answers to the most commonly asked questions concerning the issue of compulsory health insurance. Armed with this material, a good speaker will have little trouble making a splendid, positive case before even a difficult group.

ENDORSEMENTS . . . PROCEDURE

Since the value of formal action from any group is in exact proportion to the work done to capitalize on it, these are some of the things which must be done, once a good resolution is in your hands:

If it is from a strong, statewide organization you should send copies of the resolution first thing to:

Your two United States senators
Your congressmen
Your state legislators
The A.M.A. office at 1302 18th St. N.W., Washington, D. C.
The National Campaign Headquarters, 1 North La Salle, Chicago.

Every county medical society, immediately it has acted, should send copies of its resolution to:

Its two United States senators
Congressmen from its own district
State legislators from its own district
Its state campaign chairmen at the state medical association office
The A.M.A. Office at 1302 18th St., N. W., Washington, D. C.
The National Campaign Headquarters, 1 North La Salle, Chicago.

Action of other county or city organizations should be reported to United States senators only when deemed of sufficient importance to merit such handling, but should be reported at once to all others listed above.

Resolutions sent to all congressmen should be accompanied by covering letters asking for a reply, in order to keep advised, if possible, of the position of your legislative representatives.

Originals of all resolutions should be kept in the originating office, unless otherwise requested.

Copies of resolutions should reach the press and radio on the same day action is taken if possible, through your state or county publicity channels, as determined by your campaign chairman.

When an organization has acted, it should really be asked to go to work in the campaign:

a. Getting literature to its membership, either through meetings or by use of its mailing list, or both.

b. Using its house organ or news letter for both news and editorials on the issue.

c. Offering its talented members as volunteer speakers on the issue of compulsory health insurance.

d. (Members on record in one organization can help, too, in presenting resolutions to other organizations of which they are members, and helping to steer them to favorable conclusions.)

Most of the state medical associations have working organizations long in existence and thoroughly ready and able to handle their part in the campaign.

6. Machinery will have to be set up in the few states where it is not already operating, probably under the direction of the state society office, to see that shipments of materials from the national headquarters actually get into doctors' offices and finally into the hands of doctors' patients.

It is important to name the person, presumably in your state association office, to whom the national headquarters will channel supplies of literature and other materials for fast distribution in your state. His name, address, and telephone number should be sent at once to the national campaign headquarters.

In some states it may be desired that supplies go direct to the county society offices. However, it is the feeling of the national campaign directors that campaign materials in general should channel through the state medical association machinery, and down to the county societies under the directives of the state itself. That, we believe, should be determined within each state.

AUXILIARIES ARE EFFECTIVE

Obviously, the women's auxiliaries of the medical societies will be able to carry a big share of the load. As some of the California representatives will tell you, in that state's campaign on the issue of compulsory health insurance the women accomplished some of the most effective work done in speaking before women's organizations, in literature distribution, in securing endorsements and in keeping the club and women's editors enthusiastic on the issue.

COMMITTEES

It is important within the states to organize committees of doctors, both on the state and county basis, who are able to give their time to the campaign objectives, to handling speaking engagements and to working on endorsements.

Laymen may be added to committees as time goes along, as the work gets heavier and as laymen become more interested in the issue. (Committees should be kept close-knit, however, and controlled by the profession's own chairman.) In any case, help from leaders outside the medical profession should be sought and welcomed. Not only will such aid lighten the load on doctors, but it will help build the broad public picture of the danger in socialized medicine to every element of our national life.

CONCLUSION

We recognize that A.M.A.'s permanent staff, headed by Dr. Lull, has a full load of work in just maintaining its normal activities. We will need a tremendous amount of help from them, however, and we have had many warm assurances of their desire to be in the front lines of the battle, as they have been for years past.

We visualize that they will serve in many capacities, giving the national campaign the aid of their counsel and guidance, as required, providing the background material and the knowledge of the vast medical organization which we so badly need, flying into key states to carry the message, maintaining lines of communication with all the state and county societies, opening doors everywhere that are vital to the success of medicine's campaign.

The Public Relations Department of the A.M.A., under the very capable hands of Larry Rember and his staff, is going to have a tremendous task to perform. Its work will be vastly heavier as a direct result of the campaign. The states, incidentally, probably will see more of Mr. Rember, Mr. Bach and Mr. Doscher than ever in the past, for part of their work will be "trouble-shooting" in areas where the campaign at one time or another may bog down and require a jet-propulsion assist.

Their work will coordinate closely with the national campaign and in many respects will overlap it.

They will make an intensified, affirmative campaign to drive home to the public the vital part the medical profession plays in the lives and the health of Americans. They will intensify their efforts to interpret factually—and dramatically—the work of the departments of the A.M.A.—work that daily reflects highest credit on the profession and which has made the entire world deeply respectful of the American Medical Association.

We are confident that the campaign which has been laid out is a practical, workable, effective campaign, and will produce the results we must have. We are confident that by working together we can win the fight against government-controlled medicine—and that when the fight is over, medicine will have pointed the way for the whole nation, at a time when the nation might easily travel either road—toward a controlled economy or toward a free economy.

We sincerely believe that the individual doctors throughout the nation, who have paid $25 each to tell their story to America, will feel proud, as the story unfolds toward its conclusion—proud of their part in writing one of the greatest and most significant sagas of American history.

In Memoriam

GUILFOIL, JAMES ANTHONY. Died in San Francisco, March 7, 1949, aged 61, of a heart attack. Graduate of the University of Michigan Medical School, Ann Arbor, 1911. Licensed in California in 1912. Dr. Guilfoil was a member of the San Francisco County Medical Association, the California Medical Association, and the American Medical Association.

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KAY, MILTON M. Died in National City, November 16, 1948, aged 76, of sprue. Graduate of Northwestern University Medical School, Chicago, 1905. Licensed in California in 1905. Dr. Kay was a member of the Kern County Medical Society, the California Medical Association, and the American Medical Association.

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NORTON, FRANK L. Died in Los Angeles, March 14, 1949, aged 82. Graduate of the University of Southern California Medical School, Los Angeles, 1901. Licensed in California in 1901. Dr. Norton was a retired member of the Los Angeles County Medical Association and the California Medical Association.

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SIPPY, JOHN J. Died in Stockton, March 15, 1949, aged 69, of chronic myocarditis. Graduate of St. Louis College of Physicians and Surgeons, 1899. Licensed in California in 1923. Dr. Sippy was a member of the San Joaquin County Medical Society, the California Medical Association, and a Fellow of the American Medical Association.

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VERCELLINI, CHARLES H. E. Died in Los Angeles, December 22, 1948, aged 70. Graduate of Regià Universita di Torino. Facoltà di Medicina e Chirurgia, 1901. Licensed in California in 1945. Dr. Vercellini was a member of the Los Angeles County Medical Association, the California Medical Association, and the American Medical Association.

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WOLFE, HOMER HOLSINGER. Died in Fort Bragg, March 3, 1949, aged 65, of a heart attack. Graduate of Cooper Medical College, San Francisco, 1909. Licensed in California in 1909. Dr. Wolfe was a member of the Mendocino-Lake County Medical Society, the California Medical Association, and a Fellow of the American Medical Association.

John Johnson Sippy

John Johnson Sippy died in Stockton, California, March 15, 1949, after several months of failing health. Doctor Sippy was born in Venice, Illinois, July 13, 1879, and became one of America's most outstanding public health administrators. Graduating from the St. Louis College of Physicians and Surgeons at the age of 20, he located his practice in Belle Plain, Kansas. He continued this general practice for 14 years, the last five of which he also served as the part-time public health officer of Sumner County. The year after this appointment he was elected secretary of the Kansas State Health Officers Association, a post in which he continued until he left Kansas in 1919. This work in public health became increasingly challenging to him and in 1913 he dedicated his career to full-time public health work. He became epidemiologist for the Kansas State Health Department and in 1919 moved to Montana as epidemiologist for that state. In 1922 he became Montana's director of child welfare.

In 1923, as a result of serious diphtheria epidemics, San Joaquin County organized the first local health district in California. To head this unique organization, its board of trustees sought a man of vision and pioneering spirit. No more fortunate choice could have been made than John Sippy. During the next quarter century he made that local health district preeminent in our nation, both in health and in principle. He stood staunchly for grass-roots principles, local responsibility. He commanded the respect and admiration of his medical colleagues, serving as president of the San Joaquin County Medical Society for two full terms. Recognition was given him by his fellow public health specialists in the form of election to many important positions, and from 1944 to 1946 he occupied the highest elective position in American public health, the presidency of the American Public Health Association.

John Sippy recognized the responsibility of public health leaders in sharing their knowledge. From 1916 to 1919 he was lecturer in communicable diseases at the University of Kansas. In 1934 he became associate clinical professor of public health and preventive medicine at Stanford University School of Medicine. Despite the distance from his own community, he journeyed regularly to San Francisco in the teaching of medical students and to enrich their appreciation of the worth of full-time health departments and health officers. Quiet and unassuming, he was as beloved as a father or brother. But he was known, too, as utterly fearless. He cherished the highest ideals of medicine with uncompromising integrity. The inspiration he left is as priceless as the thousands of lives he saved.

Frederic Morris Loomis

Through the printed word, Frederic Morris Loomis, physician and author who died last February at the age of 71, was renowned throughout the world, but nowise and nowhere was he more favorably known than as a physician in the circle of his colleagues and his patients.

He achieved the degree Doctor of Medicine in the fact of greater than usual obstacles and later in life than most students. Dr. Loomis recorded in his writings that he knew, even in his early years, that most of all he wanted to be a physician. Lack of money forced him to relinquish a premedical education before it was well begun. To supply the immediate need he went to Alaska where he worked as a miner more years than he had intended, then for a time as editor of a newspaper in Ketchikan. But the call to medicine was strong and he returned at last to the University of Michigan. He was grad-

uated from the school of medicine there in 1912, a doctor only beginning his career at the age of 35. After four years as an instructor in obstetrics and gynecology at the university medical school, he moved to Oakland, California, and there established himself in private practice.

The glow of the ideal of physician-patient relationship was reflected ultimately in two of Dr. Loomis' best-known books, first in "Consultation Room," and then in "The Bond Between Us," written in the reflective years after he had retired from practice.

A measure of the place he held in the esteem of those who knew him best is given by Dr. Fletcher Taylor, writing in the *Bulletin of the Alameda County Medical Association:*

"His fame as a man of the world will take care of itself. His fame with us is personal. We are the privileged who knew the man who was a miner in order to become a doctor, and a doctor before he became a writer.

"So we are saddened for ourselves in this fateful loss and we truly grieve with his family, so effective in their adoring efforts in his behalf. And though we mourn, there will always live for us the inspiration of the life and works and friendship of Frederic Morris Loomis."

NEWS and NOTES

NATIONAL • STATE • COUNTY

ALAMEDA

Herrick Memorial Hospital of Berkeley has been **approved for training of residents** in pathology by the American Medical Association's Council of Medical Education and Hospitals and the American Board of Pathology, according to a recent announcement by the hospital. This is the fourth resident training program given official approval, the hospital announced, the others being surgery, obstetrics and gynecology, and psychiatry.

LOS ANGELES

Dr. Lewis E. Scharf last month was appointed acting health officer in charge of the Glendale district office of the Los Angeles County Health Department. Dr. Scharf takes the place of **Dr. Myron W. Husband,** who resigned to accept appointment as health officer of Humboldt County.

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Cooperating with the Los Angeles County Medical Association, the Los Angeles County Tuberculosis and Health Association is now conducting a six-month demonstration **program to encourage use of the chest x-ray** by physicians. The program, which opened April 18, has two purposes: the finding of unsuspected cases of tuberculosis, heart disease and other chest abnormalities, and the education of physicians in the routine use of the chest x-ray.

X-ray films are made only when the patient is referred to the unit on one of the prescription forms which were furnished each member of the Medical Association by the tuberculosis association. Patients are not accepted if they are being treated for chest symptoms for which an x-ray would normally be ordered, since the program calls for examination of those for whom a film would not be required in the routine course of diagnosis. Each film is read separately by a qualified radiologist and a chest specialist and the findings are mailed to the physician making the referral.

The free service is **limited to members of the Medical Association** or those qualified for membership. The 70 mm. portable minifilm unit is housed in Medical Association quarters for the demonstration period.

PLUMAS

Dr. Paul A. Lum, formerly in general practice in Contra Costa County, has been appointed county physician of Plumas County and superintendent of the county hospital at Quincy.

SACRAMENTO

Dr. Louis Jones of Roseville has been appointed by Governor Warren to the State Board of Medical Examiners, succeeding **Dr. Richard O. Bullis,** Los Angeles, resigned. Dr. Jones was appointed for the term ending January 15, 1953, and **Dr. Frederick N. Scatena** of Sacramento, incumbent secretary of the board, was reappointed for a similar term.

SAN FRANCISCO

Grants-in-aid to support **research in the field of heart disease** were awarded recently by the Insurance Medical Research Fund to four physicians at the University of California Medical School, two at Stanford University School of Medicine and one at Mt. Zion Hospital, San Francisco. The awards were as follows:

Dr. David M. Greenberg, University of California Medical School, for research on metabolic changes in the kidneys and arteries associated with the development of hypertension and arteriosclerosis, $15,750.

Dr. John H. Lawrence and Dr. William J. Kerr, University of California Medical School, for research on vascular physiology and disease, with special reference to total body water, $13,650.

Dr. I. L. Shaikoff, University of California Medical School, for research on the metabolic characteristics of arterial tissues and the pathogenesis of arteriosclerosis, $8,400.

Dr. J. M. Crimson, Stanford University Medical School, for research on capillary blood flow, $12,600.

Dr. Emile Holman, Stanford University Medical School, for research on experimental valvular disease, cardiac anastomoses and hemodynamics, $5,250.

Dr. Meyer Friedman, Mount Zion Hospital, for research on the distribution and fate of digitalis glucosides, $16,800.

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The University of California Medical School, University Extension, has announced two additional **postgraduate courses for the summer months,** one a five-day course in endocrinology, the other a six-day course in the medical aspects of nuclear energy. The former will be held June 20 through June 24 and the latter August 29 through September 3.

Further information may be had from Stacy R. Mettier, M.D., head of postgraduate instruction, Medical Extension, University of California Medical Center, San Francisco 22.

GENERAL

A new film, titled **"Cancer: The Problem of Early Diagnosis,"** which has received the approval of the American Medical Association's Committee on Medical Motion Pictures, has been made available to the medical profession through more than 50 state and regional distributing points. Through the efforts of its co-sponsors, the American Cancer Society and the National Cancer Institute of the United States Public Health Service, prints for single showings may be borrowed from state cancer society offices, state health departments, and four regional offices of Association Films located in New York City, Chicago, Dallas, and San Francisco.

The film, designed for general practitioners, is based on the premise that if cancer were diagnosed early and effectively treated the death rate might be reduced by almost 50 per cent.

* * *

Two California medical schools were among recipients of **grants-in-aid of research** announced recently by the American Heart Association. One grant went to the University of California School of Medicine for studies of the effect of drugs on cardiac metabolism, under the supervision of Dr. C. H. Thienes; the other to Stanford University School of Medicine for study of edema in congestive heart failure and the effects of diuretic agents, under the supervision of Dr. David A. Rytand.

Dr. Lawrence Kolb, medical director of California state hospitals, last month was appointed to serve as acting director of the State Department of Mental Hygiene to fill the vacancy caused when Mrs. Dora Shaw Heffner resigned the post because of ill health. Dr. Kolb was appointed to serve until a committee composed of the deans of California's five medical schools, which was appointed by Governor Warren to find "the most competent administrator available" for the post, finishes its survey and brings in its recommendation.

* * *

The first meeting of the newly formed **International Academy of Proctology** will be held in Atlantic City, N. J., June 10, 1949. The scientific portion of the program will consist of the presentation of papers and motion picture films of interest to all physicians as well as to those specializing in proctology. Further information and a copy of the program may be obtained by writing to Dr. Alfred J. Cantor, International Academy of Proctology, 43-55 Kissena Blvd., Flushing, N. Y.

* * *

Establishment of a **National Institute of Mental Health** to intensify efforts toward the prevention, control and treatment of mental illness was announced recently by Surgeon General Leonard A. Scheele of the U. S. Public Health Service. Research will be carried on outside of the Public Health Service through research grants and fellowships awarded to non-Federal research institutions and individual scientists. Training of personnel and the establishment of community mental health facilities also will be assisted through grants-in-aid.

Despite the nation-wide shortage of professional personnel, mental health programs have been initiated in 27 states and territories and have been expanded in 24 others since passage of the Mental Health Act.

* * *

At a recent convention in San Francisco of the **California Society of Anesthesiologists,** which was formed a year ago as a merger of similar organizations in Northern and Southern California, Dr. Forrest E. Leffingwell of San Marino was elected president, succeeding Dr. Charles F. McCuskey of Los Angeles. Dr. Fenimore Davis of Oakland succeeded Dr. Thorne Hopkins of Fresno as vice-president, and Dr. Bruce M. Anderson of Oakland was reelected secretary.

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The Fifth National Conference of County Medical Society Officers (Grass Roots Conference) will be held in Atlantic City, Sunday, June 5. This is the day prior to the opening of the A.M.A. House of Delegates. Morning and evening sessions will be held. The morning session will be devoted to specific county medical society problems, with panel discussions on: (1) The problem of emergency calls; (2) Indigent medical care plans; (3) The National Education Campaign. The last hour of the morning session will be given over to questions on the National Education Campaign, with Mr. Clem Whitaker and Miss Leone Baxter, campaign directors, present to provide the answers. The evening session, which will be open to all physicians and their wives, will feature talks by Mr. Whitaker and the Honorable John L. McClellan, U. S. Senator from Arkansas.

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Examinations of candidates for the position of **Chief of the Bureau of Hospitals** in the California Department of Public Health will be held June 25, 1949, by the State Personnel Board. The deadline for filing of applications to take the examination is June 4. Examinations for the position, which carries with it a salary range of $644 to $782 a month, will be held in Sacramento, San Francisco, Los Angeles and in "such other places in California and other states as the numbers of candidates warrant and conditions permit."

Scheduled for June 21, 1949, are examinations of applicants for the position of **Assistant Medical Director,** Industrial Accident Commission, for which the salary range is $530 to $644 a month. The closing date for the filing of applications is May 31, and the places of examination are the same as those designated for the candidates for Chief of the Bureau of Hospitals.

Full description of the requirements for both positions may be had from the State Personnel Board in Sacramento, San Francisco and Los Angeles, or at any local Department of Employment office.

Discussion of **compulsory plans for medical care** and for disability compensation will highlight the fifth annual meeting of the Conference of Presidents and officers of State Medical Associations to be held at Atlantic City on Sunday afternoon, June 5, the day preceding the opening of the A.M.A. general sessions. The meeting will be open to all physicians.

Cecil Palmer, English publisher, author, and journalist, will tell of the impact of socialized medicine on the British doctor and his patients. Palmer, now completing a tour of America, has been a brilliant spokesman for the British Society for Individual Freedom. An American viewpoint of the British health system will be given by W. Alan Richardson, editor of *Medical Economics,* who recently went to England for a first-hand study of all phases of the program.

With compulsory disability compensation programs operating in three states, and Washington and New York the latest to pass such laws, the conference presents two speakers on this vital question. Edward H. O'Connor, managing director of the Insurance Economics Society of America, will discuss the legislations, and Dr. Bert S. Thomas, medical director of the California program, will tell of the medical implications of cash sickness compensation acts.

The A.M.A. relationship to the state societies will be reviewed by Dr. George F. Lull, secretary of the A.M.A. and The Problems Facing the State Association at the Crossroads will be the subject of a talk by Dr. Clarence Northcutt, president of the Oklahoma State Medical Association. Plans are pending for the presentation of views on national health legislation by a member of Congress.

INFORMATION

Department of Public Health

Bureau of Food and Drug Inspections Statement Concerning Lithium Chloride

When lithium chloride was first proposed for use as a salt substitute, the Food and Drug Administration started pharmacological experiments to determine its safety and recommended that manufacturers likewise check the safety of the product before marketing it. Experiments in our laboratories recently showed that lithium chloride is toxic to animals. However, a large manufacturer of a lithium chloride-containing salt substitute reported that extensive clinical experiments sponsored by him showed lithium chloride to be safe for human beings, thus casting some doubt upon the applicability of animal experiments to humans.

Recently, untoward reactions in humans consuming Westsal which may have been due to lithium chloride came to our attention, and recall of lithium chloride preparations was suggested to and inaugurated by manufacturers.

On Friday, February 18, the Food and Drug Administration learned of deaths attributed to lithium chloride and public announcements of the dangers of lithium chloride were distributed immediately.

Inquiries from consumers show that salt substitutes containing lithium chloride were being used by many individuals on the advice of doctors who understood that the substitutes were harmless. At the time of the radio and newspaper publicity, these consumers in large measure had not heard of the dangers accompanying use of lithium chloride.

Consumers who have been using a lithium chloride-containing salt substitute should discontinue use immediately. If symptoms of lithium poisoning have not appeared, it is likely there will be no damage. Medical reports show that people who suffered mild symptoms of poisoning recovered when they discontinued use of lithium chloride.

In case of severe symptoms, the physician should be consulted immediately. It has been suggested that cautious intravenous administration of sodium chloride solution may be of value in treating poisoning from lithium chloride.

The symptoms of lithium chloride poisoning are drowsiness, weakness, loss of appetite, nausea, tremors of the extremities, blurring of vision and coma. Products containing lithium chloride are usually sold in drug stores and/or health food stores, and every effort should be made to remove these products from the market, notifying the dealer to contact the wholesaler and/or distributor from whom these products were purchased so that they may be returned for credit.

The brands on the California market containing lithium chloride as an ingredient are:

Westsal and *Wes-sal*—Manufactured by Westwood Pharmaceuticals, Division of Foster Milburn, Buffalo, New York.

Milosal—Louis Milani Foods, Inc., 4058 South Walker Avenue, Maywood, California.

Foodsal—Foods Plus, Inc., New York, N. Y.

Saltisal and *Salti-salt*—Lueth's Bakery, Kansas City, Missouri.

Neocurtasal—Winthrop Chemical Company, New York, N. Y. *Only Neocurtasal which is sold to food industries such as bakery and cracker manufacturers in containers of 25 pounds or larger, said product containing lithium chloride.* Note: Neocurtasal sold over the counter and on prescription and in health food stores, which contains potassium chloride, ammonium chloride, starch, potassium formate, calcium formate, and magnesium citrate, is not involved in this investigation and should not be removed from the market.

Attention should also be called to the fact that there may be other salt substitute products on the market containing lithium chloride.

It has come to our attention that some consumers believe some brands of ordinary table salt are causing these injuries. Lithium chloride is marketed as a liquid. The granular table salt, sodium chloride, is not under suspicion.

Hospitals, drug stores, dealers and health food stores should be instructed to return the above brands of salt substitutes containing lithium chloride to the distributor, obtaining a receipt for the same.

Dried Plasma Surplus Exhausted

The State Department of Public Health has been advised by the American Red Cross that the surplus supply of dried plasma is now exhausted and that the distribution by the Red Cross to the civilian medical practice has been discontinued. When current stocks in health departments and hospitals have been used the State Department of Public Health will therefore not be able to replenish these stocks.

In October 1946 the American Red Cross made this biologic surplus to the need of the armed forces available to the civilian physicians. The Division of Laboratories of the State Department of Public Health undertook the distribution to the local health officers who in turn dispensed the plasma to their local physicians. Since the inauguration of the program over 70,000 units have been distributed.

Damage Action Against Municipality Based on Poliomyelitis Infection From Sewage

In trial of an action for damages of $100,000 brought against the City of Mill Valley, Marin County, by the parents of a minor child who was alleged to have contracted poliomyelitis from sewage that backed up into the child's residence, the jury brought in a verdict in favor of the defendant municipality. The suit is believed to be the first in California based on an allegation that the disease was caused by exposure to sewage.

As the child in whose name the action was brought was one of 11 persons in Mill Valley who had the disease during the epidemic of 1945, the trial was seen as one of far-reaching implications, and appeal by the municipality in event the verdict had been unfavorable was considered a certainty.

Judge Edward Butler, who presided over the Marin County Superior Court in which the case was tried, had denied a motion by the defendant that the action be dismissed for lack of evidence and precedent. In denying the motion the court ruled that the case was of such importance as a test that it should be decided by a jury.

There was testimony for the plaintiff that on several occasions sewage had backed up into the basement of his residence and that after the floor was cleaned he had played with toys there. It was contended that as a result he had contracted poliomyelitis May 26, 1945. Testimony as to whether the sewage had spread upon the basement floor in the last week in April or, as the city contended, in January and February of 1945, was conflicting. Evidence was also introduced that fecal matter was seen on the streets and in an adjacent creek, but the exact time was not shown, nor the proximate cause.

Several clinicians testified for the plaintiff and three epidemiologists and clinicians for the defense. Among the expert witnesses for the defense was Dr. William McD. Hammond, professor of epidemiology at the University of California Medical School. Dr. Hammond had studied the Mill Valley outbreak from the start, observing patients clinically, obtaining histories and securing virus specimens. His testimony, in effect, ruled out sewage as the source of infection and gave person-to-person spread, directly or indirectly, as the most likely cause.

Plaintiff's motion for a new trial was denied.

BOOK REVIEWS

TREATISE ON SURGICAL INFECTIONS. By Frank Lamont Meleney, M.D., Associate Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University; Associate Visiting Surgeon, Presbyterian Hospital, New York. Oxford Medical Publications, Oxford University Press, New York, 1948. $12.00.

This volume represents the observation of a keen student of both bacteriology and surgery over a busy 25 years of active practice. As the author mentions in the preface, the advent of the sulfonamides in 1935 and later World War II postponed the publication of this work and, of course, greatly changed and augmented this study.

The purposes of the book, as stated by the author, are threefold: (1) To maintain high standards of sterile technique; (2) to elucidate the bacteriological problems of surgery; and (3) to describe the natural course of surgical infections with and without surgical procedure and their therapeutic aids.

Of interest is a foreword written in 1940 by Hans Zinsser, who read the first draft of the manuscript.

The first eight chapters are devoted to the study and maintenance of sterile technique. The next four carry out the second purpose of the book, namely the elucidation of bacteriological problems. The remainder of the work is devoted to the third purpose, as outlined above.

Of particular interest are the chapters on the uses of zinc peroxide and bacteriophage in active therapy of infection. The latter is chiefly of academic interest, and at least to date there is but little clinical application that is feasible.

As might be expected because of the rapidly changing picture of antibiotic therapy, the last chapter devoted to this subject is the least up-to-date and probably even misleading. Two cases are cited, one of anaerobic cellulitis of the arm and one of gas gangrene of the abdominal wall, in which the patient died despite penicillin and surgical incision. It should be noted that the total dosages of penicillin amounted to 280,000 units and 175,000 units, respectively. These doses are known now to be far too small, and in no way should these cases be used to illustrate the questionable value of penicillin in anaerobic infection.

The value of such a volume to the individual surgeon is of some question. Its place will probably be as a reference book in a well-equipped medical school library or the libraries of the larger hospitals.

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THE PHYSIOLOGY OF THE EYE. By Hugh Davson, D.Sc. (Lond.), Honorary Research Associate, University College, London. 301 Illustrations. The Blakiston Company, Philadelphia, Pa., 1949.

This book, written by a former Canadian professor of physiology, who has had experience in teaching in ophthalmology, fills a long-felt need.

Following a description of the anatomy of the eye the book is divided into five sections which have the following titles: Intraocular Dynamics and the Transparent Tissues; The Mechanism of Vision; The Muscular Mechanisms; Visual Perception; and Optics.

The sections discuss the important parts of the subject without overwhelming the student with too much theory or too much detail. One of the subjects that has always been troublesome to the student of ophthalmology has been optics. As the author states, "The ophthalmologist is not concerned with formulae and still less with their mathematical derivation; he must understand sufficient of the general principles to enable him to speak intelligibly on the subject to the optician." With this in mind the author has cut the formulae to a minimum and illustrates the text with clear, easily-understood diagrams.

The book is an excellent one and has already been adopted as the textbook on physiology in one of our well-known postgraduate courses in ophthalmology.

The format is excellent, the print being very readable and the illustrations being clean-cut and well printed. The book was printed in Great Britain and bound in this country.

The book can be recommended as an excellent text on the physiology of the eye for the student of ophthalmology, particularly the resident in ophthalmology.

* * *

PSYCHODYNAMICS AND THE ALLERGIC PATIENT. By Harold A. Abramson, M.D., Associate Physician for Allergy, The Mount Sinai Hospital, New York, N. Y. A Panel Discussion. The Bruce Publishing Company, St. Paul and Minneapolis, 1948, for the American College of Allergists, 423 LaSalle Medical Bldg., Minneapolis 2, Minn.

Dr. Abramson has combined in an 85-page book two of his articles on the psychologic aspects of allergic manifestations, with a panel discussion by a number of internists, allergists and psychiatrists. The author "hopes that this first step in the coordination of organizational allergy and psychodynamics will lead to the ultimate recognition of the importance of emotional factors in the routine therapy of the allergic patient by both the allergist and the general practitioner."

In the first section, Dr. Abramson reveals briefly the early psychologic explanations for asthma and rhinitis by quotations from various authors from Hippocrates to Mackenzie in 1887 and Thorowgood in 1879.

In the second section the author finds himself at a loss to always explain or completely correlate his patients' symptoms by use of the immunologic model. He thus gives brief summaries from his patients' records in which he believes factors of emotion seem to play a major part. It is emphasized by Dr. Frank Fremont-Smith that the author has every right to do this, as he has gained respectability by having done important work on physicochemical mechanisms connected with immunologic processes in allergy.

In the panel discussion Drs. Frank Fremont-Smith, Edward Weiss, Hal Davison, Sandor Rado, O. Spurgeon English, M. Murray Peshkin, John A. P. Millet and John H. Mitchell ably discuss the importance of psychodynamic factors. They are in agreement that these play important roles in the symptoms of asthma, rhinitis and urticaria.

Drs. Leon Unger and George L. Waldbott commented on the dangers of attributing all unknows to psychic factors while Rudolf L. Baer, principally interested in allergic dermatoses, stated that he had never seen a case of urticaria, atopic dermatitis or eczema in which a psychic factor could have been a major cause. He did admit that psychic factors may contribute through the autonomic nervous system.

It is admirable that several of the speakers had the temerity to admit that actually little is known, for example, about the detail of the mechanisms involved in asthma on the one hand and the neuroses on the other.

Dr. Smith brought out that the very respectable physicists no longer think of single causality and Dr. Abramson concludes that allergy and psychiatry should now be synthesized by the clinician in theory and practice.

In the opinion of the reviewer this book should be read with respect and interest by those dealing with these syndromes, but because of the multiple factors involved in many such problems a great humility should be practiced by both psychiatrists and others in relation to etiology.

Many of us have long ago accepted the value of psychotherapy, and a book such as this seems rather behind the times. However, its essential good is that we now may feel officially released from the restrictions implied by the frame of reference of immunology and should simply believe that if therapy by any procedure is efficiently successful, both for relief and prevention of symptoms, it should be used, even though essential explanations may eventually come through electrochemistry at the other end of the neuron.

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PSYCHIATRY IN GENERAL PRACTICE. By Melvin W. Thorner, M.D., D.Sc., Assistant Professor of Neurology, Graduate School of Medicine, University of Pennsylvania. 659 pages. W. B. Saunders Company, Philadelphia. 1948. $8.00.

This volume was written primarily for the use of general practitioners and students in order "to acquaint the internist and general practitioner with those aspects of psychiatric theory and practice which are of constant daily concern to him." After a preliminary few pages on the plan of the book, the second section deals with "The People," more or less typical kinds of psychiatric patients. There is a chapter on intelligent people, with above-average intelligence, in whom emotional problems interfere with adequate functioning, and another chapter on "Dull People," about those whose lack of average intelligence creates emotional, social and economic problems. Then follows a section on "People and Sex," with general considerations of basic sexual drives and problems arising from these. Chapter 5 presents the effects of unusual happenings like war, disease, social and economic failure on the equilibrium of people, emphasizing that catastrophe may actually lead to improvement in some and be used as rationalization for illness in others, and that the patient must be considered the product of the effect of environment upon his hereditary tendencies. Depressive reactions are discussed under the heading of "Unhappy People," and here manic-depressive psychosis and neurotic depressions are described. Mental symptoms resulting from organic disease of the brain of patients in whom alteration in brain function can be demonstrated are described in the chapters entitled, "Dementing People," and "Confused People," with case reports of patients suffering from general paresis, encephalitis, carbon monoxide poisoning, brain tumor, epilepsy, Huntington's chorea, delirium, alcohol, etc. Schizophrenic reactions and the paranoid reactions are referred to in sections on "Dreamy People," "Suspicious People," and "Queer and Twisted People," while other sections describe the psychoneuroses as "Anxious People," geriatric problems as "Older People," and pediatric problems as "The Children."

The third section of the book is a formal consideration of the methods of psychiatry, the history and psychiatric examination, the neurological and physical examination, and various laboratory procedures utilized. Simple advice on the psychiatric interview is given, followed by chapters on therapy, considering such varied approaches as chemotherapy, physiotherapy, hydrotherapy, insulin and convulsive therapy, electronarcosis and frontal lobotomy. In the chapter on psychotherapy, emphasis is given to the so-called short forms of psychotherapy applicable to psychiatric problems one meets in general practice. Throughout the whole book, detailed case reports are given, pointing up the personality of the patients treated and the therapeutic approach. An appendix lists the classification of mental disorders adopted from that approved by the Council of the American Psychiatric Association, and also quotes a report on commitment procedures prepared by the Group for the Advancement of Psychiatry.

The author has obviously and studiously avoided as much of the technical jargon of psychiatry as he could, sometimes sacrificing clarity as a result, but on the whole the volume is an honest exposition of the general kind of psychiatric problems that one meets in practice. Because of its simplicity, the physician who has had little psychiatric background and training can read it with profit.

* * *

THE DOCTOR WEARS THREE FACES. By Mary Bard. J. B. Lippincott Co., Philadelphia, April 15, 1949. $3.00.

The sister of the author of "The Egg and I" reveals a style suggesting that heredity may have some place in wit and humor. She is the wife of a physician practicing apparently in the Pacific Northwest and she writes ably on fourteen points in the thorny path of equanimity. Most physicians' wives will enjoy this book, especially those who attended the quinquennial three-ring circus known as an A.M.A. Convention in San Francisco. The chapter on the author's pursuit and capture of her husband is perhaps the most entertaining in the book. Recommended for light summer fare and not social significance.

* * *

ESSENTIALS OF GYNECOLOGIC ENDOCRINOLOGY. By Gardner M. Riley, Ph.D., Associate Professor of Obstetrics and Gynecology, University of Michigan Medical School. Caduceus Press, Box 17, Ann Arbor, Michigan, 1948. $3.00.

This is an excellent little book which should appeal to students, hospital house officers, and to practitioners who deal with women, because it is quite comprehensive yet is brief, clear and to the point. It is also well documented. The first section, containing 13 chapters on 62 pages, reviews endocrine physiology. This includes discussion of the pituitary hormones, the endocrine function of the ovaries, the effects of the ovarian hormones, the hormonology of menstruation, and the other cyclic changes in the genital tract. Also discussed in this section are the hormone changes associated with pregnancy and with the physiology of the breasts. Short sections take up the hormonal physiology of the adrenal glands, the thyroid and parathyroid glands, the pancreas, the thymus and the pineal body, particularly as their functions are related to genital function.

A second section discusses clinical aspects. This includes consideration of puberty and the menopause, menstrual dysfunctions and the endocrine disorders of pregnancy. Abnormal sexual development such as infantilism and pseudohermaphroditism are discussed. Even the endocrinology of the male is considered. The carcinogenic possibilities of the steroid hormones are discussed.

The third section takes up diagnostic procedures such as the vaginal smear, endometrial biopsy and hormone assays, as well as giving a brief exposition of hormone chemistry, and a tabulation of potent endocrine preparations which are available commercially.

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1948 YEAR BOOK OF GENERAL MEDICINE. Edited by Paul B. Beeson, M.D., J. Burns Amberson, M.D., George R. Minot, M.D., William B. Castle, M.D., Tinsley R. Harrison, M.D., and George B. Eusterman, M.D. The Year Book Publishers Inc., 304 S. Dearborn St., Chicago, 1948. $4.50.

The 1948 Year Book contains abstracts of the pertinent literature in general medicine for the last half of 1947 and the first half of 1948. Paul B. Beeson has replaced George F. Dick as the editor for Infectious Diseases and Tinsley R. Harrison has replaced William D. Stroud as editor for Diseases of the Heart and Blood Vessels. The articles are well abstracted and well arranged. The Year Books are of use essentially to the busy practitioner who can not, or will not keep up with medical literature himself and who prefers his medical information predigested. They are preferable to the summaries of the drug houses which, while often excellent, tend to be biased in favor of their own products.